1. 215
936

The New England
Journal of Medicine

VOLUME 215 AUGUST 13, 1936 NUMBER 7

THE MASSACHUSETTS CANCER PROGRAM*

BY HENRY D. CHADWICK, M.D.,† AND HERBERT L. LOMBARD, M.D.†

IN 1926 the Massachusetts cancer program was inaugurated. It was the result of work on the part of such a complete cross-section of the population of the State that it might well be studied with interest from the standpoint of an expression of State-wide and concerted opinion, even if it had no deeper significance. A physician realizing the need for radium which few could afford; the industrious father of a large family realizing that a prolonged cancer illness in his family had a deep psychological effect upon every member of his household; a Catholic priest, a student of human nature and well versed in medicine itself, recognizing the imminent need of greater facilities because of the aging population and of the effect of hopelessness general among patients with this disease and their families; a women's organization which circularized its members with literature and studied the problem and then expressed itself concertedly for the program; veterans' clubs; national groups,—all came forward and pleaded for the cancer program. There were some among the profession who questioned its place in the State Department of Public Health, but after it became law they gave their cooperation.

The program is now in its tenth year and the death records of Massachusetts for 1935 show the first decrease in cancer deaths of both sexes in the twentieth century. On seven previous occasions the cancer deaths among males have shown a decrease and on four previous occasions among females, but 1935 is the first year in which cancer deaths have decreased in both sexes simultaneously. Whether this is significant remains to be seen. Whether or not 1935 marks the beginning of a new trend in the disease, the finding of Bigelow and Lombard‡ reported in 1934, that the adjusted rates of cancer among women had been stationary from 1920 to 1932, has been upheld for three additional years and the radical change in the Massachusetts cancer program beginning October, 1934, offers hope of greater improvement in the future.

The Massachusetts cancer program as originally devised, and as still functioning with modifications, contains five major activities: hospitalization, tumor diagnostic service, research, diagnostic clinics, and education.

TABLE 1

MASSACHUSETTS AGE, SEX ADJUSTED CANCER DEATH RATES

Adjusted to Massachusetts 1900 Population

Year	Rate per 100,000 Population		
	Male	Female	Total
1920	84.5	128.6	107.1
1921	85.9	129.4	108.2
1922	83.5	126.9	105.8
1923	85.4	125.8	106.1
1924	92.2	127.5	110.3
1925	89.6	130.0	110.4
1926	94.3	127.5	111.4
1927	92.5	128.9	111.1
1928	94.3	128.8	112.0
1929	93.3	127.4	110.8
1930	96.8	125.4	111.5
1931	93.5	126.2	110.3
1932	96.7	129.2	113.4
1933	101.5	128.7	115.5
1934	107.2	129.4	118.6
1935	99.2	125.2	112.5

The Pondville Hospital opened in 1927 with a capacity of ninety beds. This institution admits patients with cancer and with suspicion of cancer, who cannot otherwise receive adequate care. In 1930 the bed capacity was increased to 115 and late in the fall of 1935 additional beds were made available, bringing the total to 145. With excellent facilities and with an exceptionally competent staff this institution was caring, in 1935, for approximately 8 per cent of the cancer load. In 1935 the Legislature authorized an additional cancer hospital in Westfield. This building will be opened in 1937. In spite of the increased facilities for hospitalization at Pondville and the additional facilities that have been made available by the new Palmer Memorial Hospital in Boston, opened in 1927, the Rose Hawthorne Lathrop Home in Fall River opened in 1932, the waiting list at Pondville indicates that at the pres-

*From the Massachusetts Department of Public Health.

‡Bigelow, G. H., and Lombard, H. L.: Change in the Massachusetts cancer trend. New Eng. J. Med. 210:526 (March 8) 1934.

†Chadwick, Henry D.—Massachusetts Commissioner of Public Health. Lombard, Herbert L.—Director, Division of Adult Hygiene, Massachusetts Department of Public Health. For records and addresses of authors see "This Week's Issue," page 303.

ent time bed facilities for this disease are inadequate.

Antedating the present cancer program by several years, a Tumor Diagnostic Service has been maintained by the Department in conjunction with the Harvard Cancer Commission. Any physician or hospital may have suspicious tissue examined to determine the presence or absence of cancer. Approximately 2500 specimens are examined yearly, and there is a steadily increasing number of inquiries from the physicians regarding the most satisfactory treatment of many types of tumors.

Research is primarily of a statistical nature and during the nine years of the program has covered questions on care and treatment of cancer patients, as well as factors dealing with the etiology of the disease.

In the early part of the program, group consultation in the clinics was suggested but was not adopted in most cases. Social service has been an integral part of the clinics from the beginning. A uniform record system giving social data was required of all clinics but medical records were inadequate. In some cities the clinic activities were divided by several small clinics being held in separate hospitals.

Educational committees were formed in the clinic cities. Their main object was to impress the public with the early signs of cancer, the danger of delay, and the use of modern facilities for care and treatment. This was done largely through newspaper articles sent to the committees by the Department. The radio was used frequently. Throughout the program special cancer days or weeks were held. Some of these were in the clinic cities and others were State-wide.

In the fall of 1934 the whole cancer program was reviewed and efforts were made to correct its shortcomings. Steps were taken to reorganize the clinics. Group consultation was made mandatory in all clinics, and cities having the clinics divided among several hospitals were urged to unite at one hospital. The three Pittsfield clinics united at the St. Luke's Hospital. The clinic at North Adams, which originally was a part of the Berkshire County clinic, separated and became the North Adams clinic. The Newton clinic was abolished due to the small attendance. The Worcester clinic, which met in six hospitals, united at the Memorial Hospital. The Worcester North clinic separated into the Fitchburg and Gardner clinics. The Springfield clinic, which had previously been in three hospitals, united at the Springfield Hospital.

The clinics were advised to become consultation clinics for the profession. Prior to this time newspaper material had advised an individual with symptoms of cancer to go to his physician or to a cancer clinic. The advice to go to a cancer clinic has been discontinued and all educational material now directs the patient to go to his physician. The physicians are advised to use the cancer clinics as consultation centers.

Uniform medical records were demanded of the clinics and special teaching clinics, at least one a year, were also required. At the teaching clinics either the local staff or a consultant brought in from some other locality gives a demonstration clinic for the profession in that locality. There has been an average of three teaching clinics a month for the past six months and present developments indicate a substantial increase in the immediate future.

Physicians are urged to go with their patients to the clinics and avail themselves of this free consultation diagnostic service. In one clinic held recently seventeen patients, all accompanied by physicians, attended.

TABLE 2

CANCER PROGRAM STATISTICS

	1927	1931	1935
Cancer deaths.	5454	5859	6482
Cancer in clinics.	305	674	1001
Precancers in clinics.	88	213	312
Others in clinics.	967	2160	2342
Number of clinics.	6	13	18
Number of towns represented.	134	224	239
Number of specimens sent to Tumor Diagnostic Service.	2550	2530	2575
Admissions to Pondville Hospital.	123	907	1186
Beds in Pondville Hospital.	90	115	145
Median duration of delay in months between first symptoms of cancer and consultation with physician.	6.8	6.4	6.1
Median duration of delay in months between visit to first physician and clinic.	5.2	5.8	3.3
Percentage of all patients sent by physicians to clinics.	20.1	38.2	58.2
Percentage of cancer patients sent by physicians to clinics.	44.8	60.0	74.0
Percentage of patients having seen only one physician before coming to clinic.	43.3	49.6	51.8

Table 2 gives the comparison of the clinic statistics for the years 1927, 1931, and 1935, together with similar figures for Pondville and the Tumor Diagnostic Service. The Tumor Diagnostic Service shows little change throughout the period. The duration of delay between first symptoms and first consultation with a physician has decreased slightly over the period but not to the extent desired. The duration of delay between the first visit to a physician and attendance at a clinic has shown a marked improvement in the last four years, but even now this delay is too great. The percentage of patients referred to clinics by physicians has in-

creased markedly both for total attendance and for cancer patients. There is less medical "shopping" as evidenced by the increased percentage of patients being referred by the first physician consulted. While the cancer deaths in the State over the nine-year period showed a 19 per cent increase both the clinic attendance and the admissions to Pondville have shown far greater increases, indicating that the use of facilities for diagnosis and treatment is increasing to a much greater extent than the disease itself.

TABLE 3

PERCENTAGE OF CANCER PATIENTS ATTENDING THE CLINICS ALIVE AT YEARLY INTERVALS FOLLOWING CLINIC ADMISSION

Number of cancer patients	5422
Percentage lost and unknown	5.6
Alive 1 year after	66.5
Alive 2 years after	53.0
Alive 3 years after	45.8
Alive 4 years after	40.8
Alive 5 years after	36.4
Alive 6 years after	32.8
Alive 7 years after	30.1
Alive 8 years after	24.3

Table 3 shows the percentage of cancer patients alive after various intervals of time following their admission to the clinics. While this table does not indicate necessarily the percentage of cures, it points toward either cure or at least prolongation of life in most of the 24.3 per cent of cancer cases attending the clinics alive at the end of eight years. An unpublished study of untreated cancer showed only 0.7 per cent of the cases alive at the end of the same interval of time.

The change in the educational work followed the reorganization of the clinics. It was recognized that the general education in cancer that had gone on for eight years was excellent because it had laid the foundation in the public mind for the acceptance of the more specific and personal instruction which would have to follow before the results of education could be really felt.

The physician must be the pivotal figure in cancer control. With this in mind, the organization in every city and town in the State of a co-operative cancer control committee was begun. Each of these committees includes representatives of all organizations in the community —social, racial, religious, fraternal, service, military, and political. Each individual member is asked to arrange with the organization that he represents for at least one meeting a year at which cancer will be discussed by one of the local physicians. A central committee is formed to take care of administrative details. An important feature stressed is that the meetings be sufficiently small to encourage free and informal discussion.

Every physician in the State has received a copy of the cancer number of *The Commonhealth* in which were thirty-one articles by specialists on various types of cancer as well as general historical and statistical material. Other literature is sent upon request. Monthly bulletins composed of abstracts of current articles on various phases of cancer are sent to physicians throughout the State.

This program is mutually instructive: the physician has to keep abreast of the subject of cancer so that he can teach and answer questions and the layman learns from the physician.

In 1935, through additional legislative appropriations, the Greenfield clinic was re-opened, clinics were established at the Beth Israel Hospital in Boston, at the Cooley Dickinson Hospital in Northampton, at the Addison Gilbert Hospital in Gloucester, and at the Anna Jaques Hospital in Newburyport. Plans are now under way for two new clinics, one at Hyannis and the other at Fall River.

The new clinics, up to the present time, are showing better attendance per population than any of the other clinics with the exception of Pondville. Practically all of the cases in the new clinics are coming from physicians.

The medical profession in Massachusetts and the State Department of Public Health have shown in this cancer program what can be accomplished by complete co-operation. In no better way can the medical profession render service and maintain its traditional standing with the public than by showing, as it has, a willingness to take advantage of the facilities provided by the State and make use of them for the benefit of patients who otherwise could not obtain the best diagnostic aids or hospital treatment.

CONVALESCENT CARE IN CHRONIC ARTHRITIS*

BY JOHN G. KUHNS, M.D.,† AND ROBERT J. JOPLIN, M.D.†

CHRONIC arthritis is a systemic disease with articular symptoms which usually develop slowly and which require a very long time for their complete subsidence. Death or rapid recovery rarely occurs. The repair of the articular lesions occurs slowly and often imperfectly. Recovery of the general health may require an even longer period of time.[1] The disabilities and deformities which often come insidiously in the wake of chronic arthritis may need many months or even years for their correction.

A long period of medical supervision is imperative in the treatment of such chronic disease in order to secure the closest possible approach to normal health and adequate function. It has been shown that chronic arthritis can be brought to quiescence slowly with the therapeutic measures at our disposal.[2] The period of convalescence differs greatly, depending upon the varying factors of fatigue, nutrition, infection, deformity, social and emotional adjustment, and many others.[3] The first subsidence of articular symptoms after prolonged rest rarely indicates a well patient. Recovery scarcely ever takes place so rapidly and completely that supervision can be discontinued in less than two years.

The provision of adequate medical supervision for the long period essential to the rehabilitation of these patients creates a serious problem. In the usual general hospital there is little if any provision for the prolonged hospitalization, for the vigilant aftercare required or for the special therapeutic measures that may be necessary. Often the difficulties of fitting into certain economic and social patterns in the face of physical limitations become apparent only after leaving the sheltered environment of the hospital. Many patients with chronic arthritis do not recover full function even after extensive surgery, and adjustments to permanent disabilities must be made.[4]

Cure in chronic arthritis, as in tuberculosis, is never assured. Quiescence throughout life is commonly found, but there is no certainty that a recrudescence of symptoms will not occur. We believe that there are a number of medical and hygienic measures which will make the continuance of this quiescence of the disease more certain. Without diligent guidance of the patient many factors can produce a recurrence of symptoms which may go on to even more serious crippling. During convalescence there should be medical supervision with an attempt to obtain continuous improvement in the general health and in articular function, at times improvement of the home environment, aid in social adjustment and vocational guidance and the encouragement of a healthy mental outlook.[5]

This study of the convalescent care given by the Robert Brigham Hospital to patients suffering from chronic arthritis shows that there has been a steady growth both in its intensiveness and the fullness of its scope. We are convinced that the task of keeping the ambulatory patient free from articular symptoms and able to carry on some useful work is the most important and the most difficult one for the physician or institution treating chronic arthritis. The end-results tabulated do not represent a true cross-section of the recovery in chronic arthritis,[6] since very few early cases have been treated. Most of the patients were sent to the Robert B. Brigham Hospital by their physicians only after they had become completely helpless. All were from the lowest economic stratum.

There were 661 patients suffering from chronic arthritis who were discharged from the wards of the hospital up to one year ago or longer. There were 423 females and 238 males; 452 were diagnosed as atrophic arthritis, 198 as hypertrophic arthritis and 11 as chronic gonorrheal arthritis.

Table 1 summarizes the condition of these patients on admission to the hospital, on discharge from the hospital and at the present time.

The average stay in the hospital was eleven and one-half months for atrophic arthritis with the average for men and women being about the same. The figures show that about two-thirds of the living patients with atrophic arthritis are working. We classed as working only those who have returned to their previous occupations or were able to maintain economic independence. The figures of the present status of patients with atrophic arthritis show no improvement from the time of discharge. This has been due in part to inadequate aftercare. There has been a decided increase in the number returned to work following discharge from the hospital in the past few years through more intensive supervision in the home and the outpatient clinic. Moreover, a number of these patients were beyond middle life when they entered the hospital, and they have many of the disabilities incident to old age. The average age for the cases of atrophic arthritis on admission to the hospital was 37 for men and 38

*From the Robert Breck Brigham Hospital.
Read in abstract at the clinical meeting of the American Society for the Study and Control of Rheumatic Diseases, May 11, 1936, at Milwaukee.

†Kuhns, John G.—Chief-of-Staff, Robert Breck Brigham Hospital. Joplin, Robert J.—Assistant Orthopedic Surgeon, Children's Hospital and Robert Breck Brigham Hospital. For records and addresses of authors see "This Week's Issue," page 303.

for women. Eleven patients with atrophic arthritis have been followed for more than twenty years, 5 of whom are now working. At times the progression of other diseases produced complete disability when the arthritis was quiescent and deformities were not serious.

Hypertrophic arthritis, as one would expect, showed a greater percentage working than atrophic arthritis. The average age on admission for this group was 57 for both men and women. Their average stay in the hospital was five and one-half months, the average stay for men being about twice that for women. There was a proportionately greater number of deaths in accordance with the usual life-expectancy in these older people.[7]

The course in chronic gonorrheal arthritis differed little from the other types of chronic arthritis. Disability in the patients not working was due more to failure in social adaptation than to physical handicap.

Table 2 is a comparison of the response of male and female patients, excluding those who have died and those whose present condition is unknown. What part sex plays in the development of or recovery from the various forms of chronic arthritis is not known. Arthritis of the spine, the so-called Strümpell-Marie type, more commonly seen in men, produces more serious permanent disability than any other type of chronic arthritis. Here early prevention of deformity is the most helpful therapeutic measure. Still's disease, the chronic arthritis of childhood, while it has progressed to severe disability in a small number of patients, shows much the same response to therapy as chronic atrophic arthritis in older persons.

The success of the convalescent care of these patients has been dependent upon a number of things. Of first importance has been the thoroughness of the supervision subsequent to discharge. For most patients, when acute symptoms have subsided and deformities, if present, have decreased so that care of the body and walking are possible, it has been found that the subsequent convalescent care can usually be carried on successfully with the patient at home. It is necessary in most cases to give full written instructions of rest periods and the amount and kind of activity permitted, dietary instructions, directions for medications and for orthopedic exercises or other physiotherapy, as well as for any apparatus worn. The hospital visiting nurse usually visits the patient at home frequently to see that instructions are being followed properly. Any difficulty that the patient is having is reported to the physician in charge of that patient. Before the patient returns to his home a careful study of the home is made by the social service. If the home is too difficult for the patient with his limited physical adaptability, a place in a convalescent home or boarding house is secured temporarily if possible or he is kept in the hospital for a longer period. Frequent visits are made by the patient to the out-patient clinic for examination and advice, and he is readmitted to the wards occasionally for laboratory tests, special medications, plaster casts and occasionally orthopedic operations. Table 3 gives a summary of the follow-up care of these 661 patients.

The greatest number of good end-results were seen in those cases where continuous supervision after discharge was possible. Without constant encouragement and direction the patients who are physically handicapped soon lose hope, they cease to co-operate, they easily become victims of nostrums and irregular practitioners, and the inevitable increasing disability which chronic arthritis produces is seen. Those who were cared for by their fam-

TABLE 1. Status of patients.

Type of Arthritis	On Admission		On Discharge		Present Time			
	Able to Work	Unable to Work	Able to Work	Unable to Work	Able to Work	Unable to Work	Dead	Cannot Be Traced
Atrophic arthritis, including Strümpell-Marie's and Still's disease	66	376	225	206 (21 died)	225	121	77	29
Hypertrophic arthritis	67	131	134	56 (8 died)	115	44	38	1
Chronic gonorrheal arthritis	0	11	8	3	7	1	1	2

TABLE 2. Response to treatment.

Type of Arthritis		Number Living	Percentage Working
Atrophic arthritis in adults	Women	225	67
	Men	95	58
Hypertrophic Arthritis	Women	95	83
	Men	64	56
Strümpell-Marie	Women	6	83
	Men	20	70
Still's Disease	Girls	17	71
	Boys	8	63

ily doctor or were under no supervison fared less well. Not all patients who refuse medical advice will eventually become bedridden invalids.[8] Spontaneous improvement does occur occasionally. The reparative forces of nature are not idle and even in the most serious cases some healing is seen. But this will progress most rapidly and completely when the patient is following an hygienic régime and when all the proper medical and orthopedic procedures are carried out to hasten recovery.[9]

TABLE 3. Supervision.

	Atrophic Arthritis		Hypertrophic Arthritis	
	Number	Working Percentage Working	Number	Working Percentage Working
Followed in outpatient service	252	77	140	81
No supervision	85	48	32	70
Cared for by family doctor	48	42	16	70
Died in hospital	21	—	8	—
Custodial care in institutions	18	—	8	—
Cared for by other hospitals	28	—	4	—

Most patients with chronic arthritis must of necessity be cared for by their family physicians. Even for the early diagnosis and treatment of these patients there are not adequate facilities for hospitalization such as there are for tuberculosis and psychiatric disturbances. Unfortunately, the family physician fails only too often in interest, in adequate knowledge or in necessary therapeutic equipment. One therapeutic procedure, either medical or physiotherapeutic, is tried to the complete exclusion of the many other immediate therapeutic needs which the patient presents. Practically nothing is done for the prevention or correction of the deformities, and increasing helplessness usually results.[10]

Another factor of great importance in the convalescent care has been the number of other diseases and disabilities found in these patients.[11] Organic disease may at times be of little significance in the treatment of chronic arthritis, at other times it may hinder or entirely prevent recovery. Almost one-half of the patients suffering from atrophic arthritis and over 90 per cent of those suffering from hypertrophic arthritis had serious organic disease, exclusive of the joints, at the time of discharge from the hospital. The most serious complication, as shown in table 4, was the rapid development of arteriosclerosis. Clinical signs as well as marked calcification in roentgenograms have been seen even in young children. These arterial changes with increasing hypertension have frequently produced serious myocardial and renal lesions.

Nephritis and myocarditis have most frequently been the additional factors producing disability in these patients. Obesity, at times associated with endocrine disturbance or faulty dietary habits, has often been found. Venereal disease has been present more often as a secondary disease than as a cause of the chronic arthritis. Mental disturbances of the less serious sort in the early stages of the disease, such as depressions and maladjustments, are the rule.[12] Occasionally these go on to full-fledged psychoses. Eight psychoses requiring institutional care were diagnosed in the hospital and 4 developed subsequent to discharge from the hospital. Manic-depressive psychoses were most frequent, with paranoia coming next in frequency. Six pregnancies occurred while these patients were being followed subsequent to discharge. In all of them the arthritic symptoms were better during the pregnancy with a more serious recrudescence, at times temporary, of the arthritis after delivery. We believe that, in most instances, active arthritis is an indication for therapeutic abortion. Two patients without any other disease that could be found besides the chronic arthritis have gone on to severe amyloidosis.

TABLE 4. Diseases complicating convalescent care.

Disease	Atrophic Arthritis	Hypertrophic Arthritis
Arteriosclerosis	41	69
Nephritis	22	9
Hypertension	19	33
Obesity	17	30
Myocarditis	16	7
Gonorrhea	12	1
Rheumatic heart disease	10	0
Psychoses	9	3
Pulmonary tuberculosis	8	2
Urinary infections	7	3
Syphilis	7	2
Pregnancy	6	0
Endometritis	6	0
Malignant neoplasms	5	6
Endocrine diseases	5	4
Diabetes	4	6
Deformities (not arthritic)	4	8
Pernicious anemia	2	1
Coronary disease	2	1

No significant difference could be found in the recovery observed in patients suffering from hypertrophic arthritis between those who came into the hospital during the first six months after symptoms had been present and those first seen late in the disease. In hyper-

trophic arthritis there is little tendency to rapid progression or to extensive deformity. Quite different were the findings in the patients with atrophic arthritis. Here, while the disease was rarely brought into quiescence rapidly, much articular damage was prevented when the patients came for treatment in the early stages of the disease. Deformities were far less extensive and severe, and this alone made the convalescent care much easier. While much can be done in the surgical rehabilitation of those who are seriously crippled, there are a number of deformities which must be considered as permanent.[13] Fifty-four of these patients were readmitted to the hospital, after the arthritis had become quiescent, for operations to improve articular function.

A large number of the patients who do not follow medical counsel or who are supervised inadequately after the chronic arthritis is apparently quiescent will have a recurrence of the arthritic symptoms.[14] Ninety-three of the cases of atrophic arthritis and thirty-six of those with hypertrophic arthritis had a relapse after freedom from articular symptoms. In many of these no definite single cause could be established. In certain cases it followed a prolonged breaking away from the hygienic régime which the patient had been following; in others it followed fatigue or long-continued strain. A few relapses occurred with the development of an acute infection. For such recrudescence of symptoms, as well as for other therapeutic procedures, there were 153 readmissions for those suffering from atrophic arthritis and 62 for those suffering from hypertrophic arthritis. As a rule prolonged hospitalization was not necessary for such recrudescence of symptoms. In a few instances, however, the disease which had recurred went on to serious permanent disability.

Death is a not unwelcome release to those seriously crippled by chronic arthritis. There were 76 deaths in those with atrophic arthritis and 38 deaths in hypertrophic arthritis. Pneumonia, myocarditis and nephritis were the usual causes of death in atrophic arthritis, as table 5 shows. In hypertrophic arthritis death was due to the causes found commonly in old age. The frequency of postoperative deaths shows that there is more than the average risk in operative procedures upon these individuals. Death in two instances was due postoperatively to fat embolism. In a small percentage of patients suffering from chronic arthritis, rapid progression of the disease and death from an intercurrent infection take place as Nissen[15] has shown. But, as a rule, once arthritis has become quiescent, there is no greater morbidity among these patients than among other members of the community.

It has long been known by students of chronic arthritis that the home environment is very important both for the development of chronic arthritis and in its influence upon the convalescence.[16] In the histories where sufficient data were available the home was of good type in 248 of the cases of atrophic arthritis and in 111 of those of hypertrophic arthritis. It was fair in 87 of the cases of atrophic and in 29 of those of hypertrophic arthritis.

TABLE 5. Causes of death.

Disease	Atrophic Arthritis	Hypertrophic Arthritis
Pneumonia	18	5
Myocarditis	13	8
Nephritis	11	2
Postoperative	6	2
Neoplasms	3	6
Blood diseases	4	1
Arteriosclerosis	4	3
Septicemia	4	0
Pulmonary tuberculosis	3	1
Urinary infections	3	2
Cerebral hemorrhage	2	2
Diabetes	1	1
Coronary disease	1	1
Endocarditis	2	0
Fracture cervical spine	1	0
Aortitis and aneurysm	0	3
Bronchiectasis	0	1
	76	38

The home was classed as unsatisfactory in 39 histories of atrophic arthritis and 10 histories of hypertrophic arthritis. In the others it was not mentioned. Much of this was inadequate social data, for many other factors are of great moment besides the location, commodiousness and cleanliness of a house. Many cases of arthritis have occurred in such homes in the face of hidden domestic friction, economic pressure, unmentioned fears and maladjustments. This has been brought much more forcibly to our attention since the onset of the depression. If the physician or the hospital can use the various social agencies available to make the home situation not only comfortable but peaceful as well, much will have been done to speed the recovery.

Only recently have we become aware of the hidden potentialities for useful service in those who are permanently handicapped. If the patient with inactive arthritis cannot be returned to his former occupation, some other may be found fitted to his limitations, but still adequate to his economic needs and his self-respect.[17] Recent legislation in a number of the states has made it possible for the handicapped to learn some other trade or calling. At the Robert B. Brigham Hospital we have used these facilities to the full extent only in the

last few years and in less than a dozen patients. Two patients with ankylosis of the spine and bilateral ankylosis of the hips were trained as draughtsmen. Two others showed an aptitude for commercial drawing. One girl with extensive deformities and ankyloses, but indomitable courage, was an honor student in an art school until her death from tuberculosis. Various placement agencies have been utilized by the social service department to find employment for the less gifted and less intelligent patients as elevator operators, night watchmen, clerks, telephone operators and so forth. Stroud[18] has well said that no country can call itself civilized which does not use its employable cripples.

This paper is no attempt to parade accomplishments in the comprehensive treatment of chronic arthritis. It is rather a statement of the many-sidedness of the problem with possible further developments in therapy. When, if ever, we shall be able to prevent chronic arthritis or bring it quickly into quiescence, then these problems will vanish. But until that time we shall have those handicapped by arthritis with us. Undoubtedly state sanitaria must be erected or community funds must be provided for the long hospitalization which many of these patients usually need. In many instances the family doctor working with established social agencies and medical consultants will find adequate facilities at his command. With our present knowledge of the causes and the therapy of chronic arthritis, while it is still incomplete, few permanently handicapped or economically unfit individuals with chronic arthritis should remain in any community.

CONCLUSIONS

1. A survey of the convalescent care of 661 patients suffering from chronic arthritis is given. A large number have been returned to economic independence.

2. The success of convalescent care has depended upon: 1. The closeness and completeness of supervision of the patient subsequent to discharge from the hospital. 2. Good treatment begun in the early stages of the disease to prevent articular damage. 3. The early recognition and treatment of complicating diseases.

3. Many factors besides the purely medical ones must be considered. The home and the social adjustment of the individual are extremely important. They alone may prevent full recovery.

4. The responsibility of the community does not end when the disease has become inactive. Training, vocational guidance and placement of those handicapped who can be employed are not only duties but are also economically profitable to the community.

REFERENCES

1. Goldthwait, J. E.: Arthritis; general considerations. Am. Med. **36**:589 (Oct.) 1930.
2. Pemberton, R., and Bach, T. F.: A brief review of the treatment of chronic arthritis. M. Clin. North America **18**:107 (July) 1934.
3. Cecil, R. L.: Influential factors in recovery from rheumatoid arthritis. Ann. Int. Med. **8**:315 (Sept.) 1934.
4. Kessler, H. H.: The Crippled and the Disabled. New York City, Columbia University Press, 1935.
5. Horwitz, A.: The cripple's place in society through the ages. Nation's Health **5**:511 (Aug.) 1923.
6. Osgood, R. B.: The most generally prevalent, the most inadequately treated, controllable chronic disease. Virginia M. Monthly **59**:573 (Jan.) 1933.
7. Kuhns, J. G.: Hypertrophic arthritis of the hip. New Eng. J. Med. **210**:1213 (June 7) 1934.
8. Pemberton, R.: Some considerations based on 300 cases of arthritis critically treated. J. Bone & Joint Surg. **17**:879 (Oct.) 1935.
9. Minot, G. R.: General aspects of the treatment of chronic arthritis. New Eng. J. Med. **208**:1285 (June 22) 1933.
10. Swaim, L. T.: Prevention of deformity in arthritis. J. Bone & Joint Surg. **18**:80 (Jan.) 1936.
11. Nissen, H. A., and Spencer, K. A.: Arthritis and systemic involvement as exemplified in a group of dead arthritics. New Eng. J. Med. **210**:147 (Jan. 18) 1934.
12. Idem: The psychogenic problem (endocrinal and metabolic) in chronic arthritis. New Eng. J. Med. **214**:576 (March 19) 1936.
13. Wilson, P. D., and Osgood, R. B.: Reconstructive surgery in chronic arthritis. New Eng. J. Med. **209**:117 (July 20) 1933.
14. Miller, J. L.: A critical review of the literature on chronic rheumatism. Arch. Int. Med. **57**:213 (Jan.) 1936.
15. Nissen, A.: Significance of life course (or level of functional activity) of the chronic arthritic. Maine M. J. **26**:181 (Dec.) 1935.
16. Guilfoil, Z. M.: Effect of economic security on mental health. Delaware State M. J. **7**:117 (May) 1935.
17. McLeod, N.: What sick and crippled men are doing for the Ford Motor Company. Mod. Hosp. **12**:1 (Jan.) 1919.
18. Stroud, W. D.: The rehabilitation and placement in industry of those handicapped with cardiovascular disease. J. A. M. A. **105**:1401 (Nov. 2) 1935.

REPORT OF A CASE OF VERTICAL FRACTURE THROUGH THE LOWER TIBIAL EPIPHYSIS DURING THE PERIOD OF BONE GROWTH AND AN OPERATION FOR THE CORRECTION OF THE RESULTANT DEFORMITY

BY ROBERT H. MORRIS, M.D.,* AND F. HAROLD DOWNING, M.D.*

VERTICAL fractures through the lower tibial epiphysis sustained during the period of bone growth, although comparatively rare, have been previously reported.[1, 2, 3] This type of fracture is an entity comparable to a Pott's or Colles' fracture. The mechanism of its production is always the same and the resultant deformity varies only in degree in each individual fracture. McFarland[3] has reported a series of twenty-three cases which came under his observation over a period of two years. Practically all of these fractures which he reports were sustained in the same way—namely, by a twist at the ankle while the foot was held firmly between the vertical iron posts of a fence. Whether the foot is held firmly or not the mechanism

*Morris, Robert H.—Assistant Orthopedic Surgeon, Children's Hospital. Downing, F. Harold—Assistant in Orthopedic Surgery, Massachusetts General Hospital. For records and addresses of authors see "This Week's Issue," page 303.

of the fracture—a violent impingement of the superior and medial border of the astragalus on the tibial epiphysis— is essentially the same. The medial and superior margin of the astragalus strikes and fractures the lower tibial epiphysis, separates the internal malleolus from the rest of the epiphysis and destroys a portion of the cells of the epiphyseal plate. The amount of separation and destruction may be great or small, according to the degree of violence of impact.

Haas[4, 5, 6] has shown in his experimental work on epiphyses and epiphyseal plates that destruction of plate cells is followed by an arrested growth. This arrested growth is general or localized according to the character of the fracture and number of plate cells destroyed. In the fracture under discussion the medial fragment (internal malleolus) can, if necessary, be replaced by means of either an open or closed reduction, but the damaged epiphyseal plate cells cannot be replaced or repaired. The crushing and destruction of the plate cells in the line of fracture cause a localized early ossification of the epiphysis with arrested growth of the medial portion of the tibia. The external portion of the tibia, as well as the fibula, continues normal growth. This type of fracture, therefore, must invariably be followed by a varus deformity of the ankle which increases as bone growth continues.

The case here reported has been followed closely for six consecutive years after the initial injury, and the changes of bone growth with the resultant deformity of the ankle, as well as the secondary deformities of the knee and spine, have been observed. The report of this case includes the history, initial treatment, postoperative treatment and an operation for the correction of the deformity of the ankle joint.

REPORT OF CASE

R. B., aged eight. While coasting, the patient ran into a tree which he struck with the sole of his right foot. He was unable to walk and was taken immediately to a hospital. X-ray films of the right ankle showed a vertical fracture of the lower tibial epiphysis with wide separation of the fragments (fig. 1).

The right ankle was manipulated and an attempt made to reduce the fracture. The ankle was immobilized in plaster of Paris. Postoperative x-ray films showed an improvement in the relationship of the fragments which was considered satisfactory (fig. 2).

One and a half years after injury: The patient reported to a clinic because of the deformity of his ankle. At this time there was marked varus of the ankle. The lower fibular epiphysis was large and prominent. He had no pain, but walked with a limp. Measurements from the anterior superior spines to the tips of the internal malleoli showed no shortening of the right leg. Measurements from the anterior superior spines to the tips of the external malleoli showed a slight increase (one quarter of an inch) in length on the right. There were no other associated deformities. X-rays were taken (fig. 3).

Two and a half years after injury: At this time the child had no pain and he could run and jump without difficulty. There was a moderate functional lumbar curve. The varus deformity was more pronounced and he had a definite limp. Measurements of the legs showed one quarter inch shortening of the medial side of the leg and an equal lengthening of the outer side. X-ray films were taken (fig. 4).

FIG. 1. Anteroposterior roentgenogram taken immediately after the injury, shows a vertical fracture of the lower tibial epiphysis with separation of the internal malleolus.

FIG. 2. Roentgenogram taken immediately after manipulation of the ankle.
This shows the medial fragment in an improved position.

Three and a half years after injury: The deformity appeared the same. The comparative measurements of the two legs were equal to those of a year previously. There was no pain and the child was able to play tennis. The lower fibular epiphysis showed increasing enlargement and there was a definite enlargement of the head of the right fibula and an outward bowing of the right knee. He said

that he "turned over" on his right ankle and that he could not always depend on it. X-ray films were taken (fig. 5).

Four and a half years after injury: At this time he had no pain and was engaging in normal activi-

FIG. 3. Anteroposterior view taken 1½ years after injury shows an area of ossification at the site of the fracture. The lower fibular epiphysis is beginning to enlarge and is slightly inverted.
The foot as a whole is in slight varus.

ties. He had sprained his ankle several times during the year. The head of the right fibula was slightly more prominent than the year before. Measurements from anterior superior spines to the

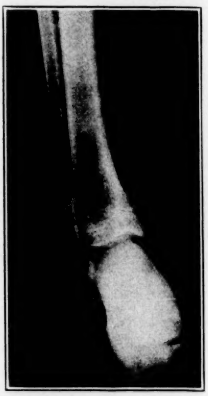

FIG. 4. Anteroposterior view taken 2 years after injury shows a further increase in deforming factor and also definite evidence of arrested growth of medial portion of tibia.

tips of the internal malleoli showed one half inch shortening on the right. Measurements from anterior superior spines to the tips of the external malleoli showed one half inch lengthening on the right. X-ray films were taken (fig. 6).

Five and a half years after injury: The patient complained that he could not depend on his ankle when attempting strenuous exercises. Comparative measurements of legs showed no change over the previous year. The head of the right fibula was

FIG. 5. Anteroposterior view taken 3½ years after injury. This roentgenogram shows further increase in area of ossification and shortening of inner portion of tibia, tilting of the ankle mortis into varus, enlargement of lower fibular epiphysis with a widening of the epiphyseal line.

much enlarged, prominent and tender. The functional scoliosis had increased markedly in the last year. Operation was advised. X-ray films were taken (fig. 7).

FIG. 6. Roentgenogram taken 4½ years after injury shows increase in all the deforming factors.

The mechanism of the fracture in this case was the violent upward thrust of the inverted astragalus against the lower tibial epiphysis which is the same mechanism as McFarland described in the twenty-three "fence fractures" he reported.

A more perfect alignment of the fragments could have been obtained by an open reduction, but such a procedure would have caused further destruction of plate cells and a resultant larger area of arrested growth in the tibia. It therefore seemed advisable to be satisfied with what was accomplished by one gentle manipulation.

FIG. 7. Roentgenogram.—5 years following injury.

The deformity was very marked and the possibility of an osteotomy was considered at the time of his visit one and a half years after injury. There was, however, no pain and the child was able to take part in all the activities of a child of his age. Operative correction of the deformity was therefore deferred until he had attained full bone growth or until such time as the deformity became a handicap to him or produced secondary deformities of the knee and spine.

Comparative measurements taken from the anterior superior spines to the tips of the malleoli showed a total growth of seven and a half inches over a period of five years. These measurements also showed one half inch shortening of the medial side of the right leg and an equal lengthening of the lateral side at the end of this period. Considering that these six years, from the age of nine to the age of fourteen, represent the period of greatest bone growth, the amount of shortening due to arrested bone growth was small and does not entirely account for the 45° varus tipping of the ankle mortise. The overgrowth of the fibula and weight bearing also played a definite part in producing the deformity. At the age of fourteen and a half years, the instability of the ankle and knee joints and the increasing deformity at the knee made it necessary to recommend operative correction of the deformity of the ankle, although it was recognized that further bone growth was to be expected.

The correction of this deformity has in the past been accomplished in several ways:

(1) A wedge osteotomy through the fibula and outer two-thirds of the tibia (McFarland). This method corrects the deformity, but leaves the affected leg shorter than the normal one.

(2) Wedge osteotomy of the tibia with obliteration of the remaining epiphyseal line of tibia and the destruction of the lower fibular epiphyseal line. This method again produces permanent shortening of the affected leg.

(3) Wedge osteotomy of the tibia with destruction of the epiphyseal line at the lower end of the fibula and tibia of both legs.

(4) Repeated linear osteotomy during the period of bone growth. This method also produces a permanent shortening of the affected leg.

The operation described below was devised and performed in an attempt to correct the deformity and provide legs of equal length.

Operation: The lower third of the right tibia was exposed by an incision four and a half inches long, parallel and lateral to the crest of the tibia. The periosteum was then incised in the line of the skin incision and separated by blunt dissection from around the tibia. About one inch above the lower epiphyseal line of the tibia the periosteum and interosseous membrane were divided horizontally. A step osteotomy was then done on the tibia, leaving three inches between the steps and the lower step about one and a half inches above the lower epiphyseal line (fig. 8A). A simple osteotomy of the right fibula was done through an incision one and a half inches long over the outer side of the leg. The upper and medial corner of the lower fragment was then removed to prevent too great pressure on the soft tissues of the leg when the foot was put into a corrected position. With a hand drill three No. 30 hardened steel pins were inserted—one through the os calcis and the other two through the upper fragment of the tibia just above the line of incision. The two upper pins were parallel to one another and at right angles to the long axis of the tibia. The lower pin was at right angles to the vertical plane of the os calcis (fig. 8B). The periosteal tube could not be sutured over the bone. The wound was closed with interrupted plain catgut for the subcutaneous tissue and black silk for the skin suture. The three pins were then fitted with a leg lengthening apparatus which was set to hold the foot in a corrected position under slight tension (fig. 8C).

Postoperative notes: The patient had considerable pain for the first three days. On the fourth day the pain subsided and lengthening was begun. Practically all the increased tension was put along the medial side of the leg so that the deformity could be overcorrected and the increased length applied to the tibia. At the end of two and a half weeks the wound was healed and the sutures were removed. Measurements showed an increase of three quarters of an inch in length of the inner side of the leg and the foot was in an overcorrected position. A long leg plaster cast was applied incorporating the pins, and the lengthening apparatus was removed (fig. 8D). Eight weeks after

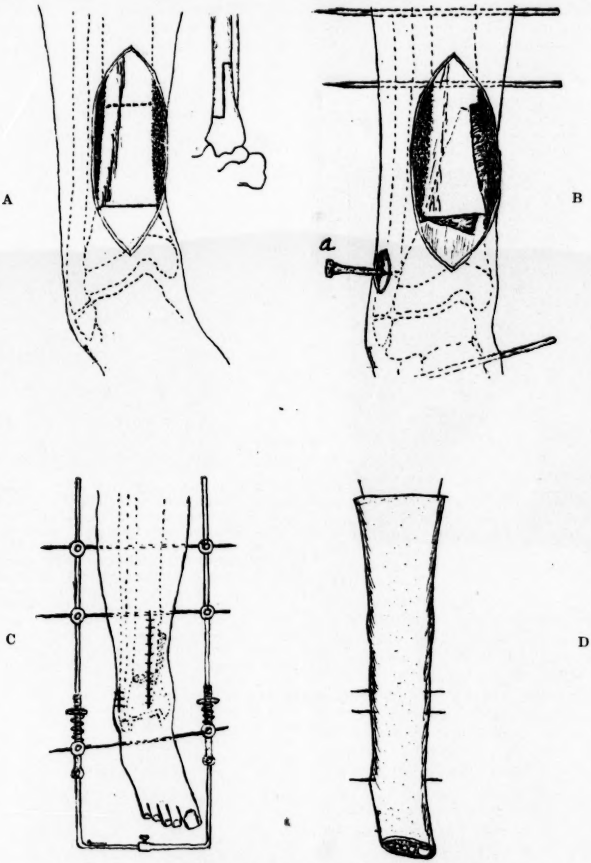

FIG. 8A. The diagram shows the anterior and posterior view of the step osteotomy of the tibia.

FIG. 8B. Osteotomy of tibia and fibula completed. Pins in place with foot partially corrected.
(a. is the osteotome in the incision through which the osteotomy of the fibula was done.)

FIG. 8C. In this diagram the lengthening apparatus has been applied to the pins.

FIG. 8D. The leg and pins are here incorporated in a long leg plaster cast.

operation the pins were removed and a short leg plaster was applied. Excellent bony union could be demonstrated both clinically and by x-ray films (fig. 9). Slight weight bearing with crutches was allowed.

At the end of twelve weeks the anterior portion of the plaster was removed and physiotherapy and active exercises were begun. This was continued for two weeks at which time he was able to discard the posterior portion of the cast and bear considerable weight with the aid of crutches without pain. A shoe with an outside raise of one quarter of an inch was then worn. By the end of six months he was bearing full weight without support. Measurements at this time showed the medial side of the right leg equal to the left and the lateral side of the right leg one quarter of an inch longer than the left. The ankle joint showed abduction 0°, adduction 20°, dorsiflexion slightly beyond a right angle and plantar flexion 30°.

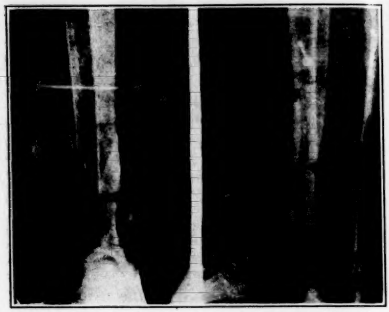

FIG. 9. Roentgenograms in the anteroposterior lateral position taken three weeks postoperatively.
The varus deformity has been corrected; there is increase in the length of the tibia. The alignment as shown in the lateral view is good.

SUMMARY

(1) The mechanism of production of vertical fractures through the lower tibial epiphysis is always the same and the invariable resultant deformity, when this fracture is sustained, varies only in degree.

(2) The slowly developing deformity is due to the arrested growth of the medial portion of the tibia. This arrested growth is the result of the destruction of epiphyseal plate cells.

(3) The initial treatment of this type of fracture is careful manipulation and immobilization. Repeated violent manipulations or open reduction for the purpose of procuring exact replacement of the fragments is contraindicated because by so doing there may be further destruction of the epiphyseal plate cells.

(4) The indication for operative measures to correct this deformity before the completion of bone growth is as follows: (a) instability of the ankle or knee or (b) an increase in the severity of the secondary deformities of the knee and spine.

(5) Previous operations for the correction of this deformity sacrifice the length of one or both legs. The operation here described corrects the deformity and maintains the normal length of the leg.

REFERENCES

1. Kimberley, A. G.: Malunited fractures affecting the ankle joint. Surg. Gynec. Obst. **62**:79 (Jan.) 1936.
2. Compere, E. L.: Growth arrest in long bones as a result of fractures that include the epiphysis. J. A. M. A. **105**:2140 (Dec. 28) 1935.
3. McFarland, B.: Traumatic arrest of epiphysial growth at the lower end of the tibia. Brit. J. Surg. **19**:78 (July) 1931.
4. Haas, S. L.: The localization of the growing point in the epiphyseal cartilage plate of bones. J. Orthop. Surg. **15**:563 (Aug.) 1917.
5. Haas, S. L.: The relation of the blood supply to the longitudinal growth of bone. J. Orthop. Surg. **15**:157 (March) 1917.
6. Haas, S. L.: Changes produced in the growing bone after injury to the epiphyseal cartilage plate. J. Orthop. Surg. **1**:67 (Feb.) 1919; **1**:166 (March) 1919; **1**:226 (April) 1919.
7. Snyder, C. H.: Deformities resulting from unilateral surgical trauma to the epiphyses. Ann. Surg. **100**:335 (Aug.) 1934.

DEHYDRATION THERAPY IN THE TOXEMIAS OF PREGNANCY

A Report of Sixty-Five Cases

BY G. ELLIOTT MAY, M.D.*

THE newer theories concerning the etiology of eclampsia and its precursor, preeclampsia, seem to indicate that the disease is of endocrine origin, possibly pituitary but more probably placental. Blood studies usually show an increase in prolan and a decrease in estrin in the toxemic patients. Local vasospasm of the terminal arterioles seems able to account for the pathological findings in the various organs and also to account for the newer concept that eclampsia is not a disease primarily of the liver or of the kidneys but rather of all the small terminal arterioles. Whether this vasospasm is local or central in origin, or both, still remains to be proved. Likewise, whether or not it is a prolan or other endocrine effect also is unsolved.

In addition to vasospasm, there is also an upset in water balance in toxemias. This disturbance in water balance which results in fluid retention in the body can be accounted for, at least in part, by arteriolar spasm, especially the glomerular arterioles of the kidneys.

Fluid retention alone probably does not account for all the symptoms of toxemia, nevertheless it does seem to produce, or at least aggravate, some of them. Edema of the body and extremities is the most obvious example of the retained fluid. Oliguria is another. Passive congestion of the kidneys produces albuminuria and can cause the appearance of red and white blood cells in the urine. Increased intracranial pressure from cerebral edema can account for hypertension, headaches, blurred vision, scotomata and finally coma and convulsions. On the other hand it is also true that localized vasospasm can account for the hypertension, and vasospasm of the retinal arterioles can cause the ocular symptoms.

Emphasizing the importance of fluid retention as a causative factor in the production of

*May, G. Elliott—Assistant Visiting Surgeon for Gynecology and Obstetrics, Boston City Hospital. For record and address of author see "This Week's Issue," page 303.

the symptoms of the eclamptic syndrome, Arnold and Fay in the August, 1932, issue of Surgery, Gynecology and Obstetrics presented a very excellent paper on the value of fluid limitation in the treatment of the pregnancy toxemias. In addition to case reports on ten patients, they gave an interesting discussion concerning the rationale of this type of therapy.

Forcing fluids on the already "water-logged" patient has seemed futile if not actually harmful, and Arnold and Fay's dehydration therapy seemed very sound in principle. Hence this series of sixty-five cases has been cared for in the manner they recommended and the results are herewith presented.

Most of the dehydrated cases presented in this series were cared for by the toxemia service of the Boston City Hospital. The rest were patients from the obstetrical service of the Newton Hospital and private patients. The control group was made up of patients cared for in the last two years at the Boston Lying-in Hospital and the Boston City Hospital.

CLASSIFICATION

In classifying these cases as preeclamptic toxemias or as mild or severe chronic nephritic toxemias, it is obvious that at times a case may have been incorrectly grouped. Too often the differential diagnosis between preeclampsia and nephritis is impossible until a subsequent pregnancy has given the kidneys the best renal function test. Realizing this, nevertheless an attempt has been made to classify the various cases and to this end, cases were considered chronic nephritis which conformed most closely to the following signs and symptoms:

1. Elevated blood nonprotein nitrogen.
2. The toxemia appearing before the fifth month of pregnancy.
3. A history of a previous pregnancy toxemia.
4. A history of previous predisposing diseases, as scarlet fever.
5. Albuminuric retinitis.
6. Moderate secondary anemia.
7. Blood pressure over 160 mm. systolic with little or no albuminuria.
8. Slowness or failure of the blood pressure and urine in returning to normal during the puerperium.
9. Impaired renal function tests.

Differentiation of the mild and severe nephritics was made arbitrarily by considering the height of blood pressure and amount of albuminuria together with the general clinical appearance of the patient and course of the disease.

TREATMENT

In caring for the outpatient cases, a very detailed toxemic history was taken, and each patient received the following printed instructions:

1. From six o'clock tonight until six o'clock tomorrow night, save, measure and record the total amount of urine that you pass. During this period do not take any more than four glasses of any kind of fluid.
2. The next day restrict your total fluid (water, tea, coffee, milk, beer, soups, fruit juices) intake to one glass less than the total urine passed.
3. Similarly each day keep track of the urine voided and during the succeeding day take one glass less of fluid, aiming always to take in less fluid than you have passed urine.
4. Keep a daily record of the urine output and fluid intake and bring this record to the clinic with you at each visit.
5. Take one or two teaspoonfuls of Epsom salts every morning so that you will have from two to four loose bowel movements each day.
6. Use no salt on your food.
7. Eat meat once a day.
8. Eat no sweets or desserts.
9. Eat four small meals a day.
10. Do not eat or drink between meals.

The treatment of the hospitalized patients was carried out along similar lines. Exceptions were, that the severe cases received no fluids at all during the first twenty-four hours and the amount of catharsis was increased. Occasionally the severe case received either 100 cc. of 50 per cent glucose solution or 20 cc. of 10 per cent magnesium sulphate solution intravenously. Protein foods were allowed in moderation. The fluid given with the Epsom salts orally was not included in the recorded intake because most of it is eliminated in the watery stools secured.

By these procedures, the level of fluid intake is placed at the point of maximum renal efficiency. There is, in addition, enough fluid in the food content to compensate for that lost in skin, breath and bowel elimination. The mild purgation assists in withdrawing tissue-bound fluid from the interstitial spaces, not only in the extremities but within the cerebral structures as well.

It has not been found necessary to continue dehydration after delivery as Arnold and Fay recommended. Their method was not used on cases admitted with actual eclampsia, because it was felt that the process of dehydrating a patient required too long a time to be of value in the convulsive case.

The control cases were treated by the old recognized methods, chiefly a low protein, salt-free diet, saline catharsis, fluids *ad lib.* or forced and sedatives.

RESULTS

a. Preeclamptic toxemias.

The first two charts show the comparison of results obtained in the dehydrated and nondehydrated cases of preeclamptic toxemia. There were twenty cases in each group. None of the dehydrated group developed eclampsia, *abruptio placentae* or macerated the fetus. All but four cases were carried to full term. The first seven cases were the most interesting and their records are given in detail subsequently. Case 8 had been controlled very well in the toxemia clinic and had been taken off dehydration. A week later she entered the hospital with a blood pressure of 160/95 mm. and an S.P.T. of albumin. Case 9 went off dehydration two weeks before term and did not return to the clinic. She later entered the hospital in labor with a blood pressure of 180/120 and a H.T. of albumin. These two cases seem to indicate the advisability of continuing dehydration in toxemias right up to the end of pregnancy in spite of any improvement shown.

In the control group one case developed eclampsia and two cases developed postpartum eclampsia. One case macerated the fetus and there was one stillbirth. Eight cases had to be induced prematurely.

CASE REPORTS

CASE 1. Mrs. L. M., a private patient, white, aged twenty-eight years, 1-para was admitted to the Baker Memorial Hospital on July 13, 1933. Her last catamenia was Nov. 25, 1932 and her expected date of confinement was Sept. 2, 1933. Her past history was irrelevant. With the exception of slight bleeding at two months her pregnancy had been uneventful. At six months an x-ray revealed a twin pregnancy. On July 12, at seven months, the patient's blood pressure was 152/98 mm.; the urine was negative, but there was slight edema of the extremities. On July 12 the blood pressure had risen to 170/100 mm., there was marked edema of face and extremities and the urine showed a trace of albumin with red and white blood cells and no casts. There were no other toxic symptoms. The patient was referred to the hospital and placed on a strict dehydration régime. Her course was as follows:

Date	Intake	Output	Blood Pressure	Albumin	Edema
July 13	0	2400 cc.	170/100 mm.	ST	++++
" 14	900 cc.	2550 "	150/90 "	VST	+++
" 15	1050 "	2250 "	130/80 "	VST	+
" 16	1050 "	2220 "	130/80 "	VST	+
" 17	1050 "	1200 "	120/80 "	ST	+

On July 18 the patient ruptured her membranes spontaneously and delivered herself of twins. Her further course in the hospital was uneventful and she was discharged home in two weeks. The twins did very well. It is of interest to note that her output for the three days following delivery totaled 6900 cc., while her intake for that time was restricted to 3600 cc.

CASE 2. Mrs. M. L., O.P.D. No. 104197, white, aged nineteen years, 1-para, was first seen in the toxemia clinic of the Boston City Hospital on Oct. 15, 1933. Her last period had been on Feb. 12, 1933 and her expected date of confinement was Nov. 19, 1933. Her pregnancy had been uneventful except for occasional headaches and vomiting.

On October 15 her blood pressure was 156/84 mm. and her urine was negative for albumin. Dehydration therapy at home was not successful, and on November 13 her blood pressure had risen to 164/96 mm. and her urine showed a slight trace of albumin. She was immediately referred into the hospital. On admission her pressure was 170/98 mm. and her urine showed a heavy trace of albumin with hyalin and granular casts and red blood cells. Her face and extremities showed moderate edema and she complained of slight headache. Her course was as follows:

Date	Intake	Output	Blood Pressure	Albumin	Edema
Nov. 13	0	?	170/98 mm.	HT	++
" 14	0	700 cc.	150/100 "	ST	++
" 15	700 cc.	800 "	140/98 "	0	+
" 16	500 "	700 "	130/80 "	0	0
" 17	500 "	750 "	120/80 "	0	0
" 18	500 "	700 "	138/98 "	0	0
" 20	500 "	720 "	140/90 "	0	0

On November 23 the patient started up in spontaneous labor and was delivered by low forceps. Her puerperium was uneventful and she and the baby were discharged in the ordinary time. Her blood pressure on discharge was 120/98 mm. and the urine showed no albumin.

CASE 3. Mrs. A. W., No. 713740, white, aged twenty-seven years, 3-para, was referred into the Boston City Hospital from the O.P.D. on June 26, 1933. Her expected date of confinement was July 11, 1933. She had had two previous normal full-term deliveries and her present pregnancy had been uneventful. She was placed on a dehydration régime and progressed as follows:

Date	Intake	Output	Blood Pressure	Albumin	Edema
June 26	0	—	170/100 mm.	ST	+++
" 27	0	400 cc.	150/110 "	SPT	++
" 28	300 cc.	500 "	140/100 "	SPT	+

Her fluids were balanced at 350 cc. intake, but her blood pressure remained around 140/90 mm. and she continued to show a slightest possible trace of albumin. Hence, on July 3 she was given castor oil and quinine, and labor ensued promptly. Normal delivery occurred on July 4. The patient had a normal puerperium and was discharged home in ten days, albumin-free and with a blood pressure of 118/80 mm.

CASE 4. Mrs. N. F., No. 713735, colored, aged thirty-three years, 4-para, was referred into the Boston City Hospital from the O.P.D. on June 26, 1933. She had had three normal full-term pregnancies. Her expected date of confinement was Aug. 19, 1933. Her present pregnancy had been uneventful except for occasional dizzy spells until June 10, at which time her blood pressure was found to be 148/104 mm. and there was slight albuminuria. She was not sent into the hospital until June 26. On admission she showed no edema or toxic symptoms. The blood chemistry was normal, and the urine sediment showed hyalin and

CHART 1

PREECLAMPTIC TOXEMIAS

Dehydrated Group

Case	Para	Age	Dehydration Started				Finished				Discharge	
			Duration of Preg. Mo.	B. P.	Alb.	Edema	B. P.	Alb.	Labor	Baby	B. P.	Alb.
				mm.			mm.					
1.	1	28	7	170/100	T	+++	120/80	VST	Sp. 7½	OK	N.	0
2.	1	19	8	170/98	HT	+++	140/90	0	Sp. FT	Twins OK	N.	0
3.	3	27	8½	170/100	ST	++	140/90	SPT	In. FT	OK	N.	0
4.	4	33	7	160/90	ST	0	140/85	SPT	Sp. FT	OK	N.	0
5.	1	27	7½	190/140	HT	+++	140/90	0	In. FT	OK	N.	ST
6.	3	35	5¼	160/90	T	+	120/70	0	Sp. FT	OK	N.	ST
7.	1	16	8	168/95	HT	+	120/70	SPT	Sp. FT	OK	N.	0
8.	3	31	8¼	156/90	SPT	+	136/88	SPT	Sp. FT	OK	N.	0
9.	1	24	7½	160/98	SPT	+	140/92	0	Sp. FT	OK	N.	0
10.	1	23	7½	144/102	0	+	130/90	0	Sp. FT	OK	N.	0
11.	1	21	7½	170/78	0	+	120/80	0	Sp. FT	OK	N.	0
12.	3	29	8½	152/90	0	+	122/72	0	Sp. FT	OK	N.	0
13.	2	24	7½	156/78	0	0	118/78	0	Sp. FT	OK	N.	0
14.	5	36	8	142/88	SPT	+	120/70	SPT	Sp. FT	OK	N.	0
15.	1	23	8	180/110	T	+	120/80	HT	In. FT	OK	N.	HT
16.	2	29	7½	175/110	HT	+++	160/100	T	In. 8½	OK	N.	ST
17.	1	24	8	148/96	HT	+++	148/88	HT	In. 8¼	OK	N.	0
18.	1	26	8	178/110	T	++	140/80	ST	In. FT	Twins OK	N.	VST
19.	1	21	8	160/100	HT	+	122/82	HT	Sp. FT	OK	N.	0
20.	2	36	7¾	140/98	T	+	120/80	T	In. 8	OK	N.	0

*Sp. FT=spontaneous full-term labor
In. 8=induced at eight months
Mac.=macerated fetus
P. P. Ec.=postpartum eclampsia
Albumin—0=none, SPT=slightest possible trace, ST=slight trace, T=trace, HT=heavy trace
Edema—0=none, +=slight, ++=moderate, +++=marked
B. P.—N=within normal limits.

CHART 2

PREECLAMPTIC TOXEMIAS

Nondehydrated Group

Case	Para	Age	Treatment Started				Finished				Discharge	
			Duration of Preg. Mo.	B. P.	Alb.	Edema	B. P.	Alb.	Labor	Baby	B. P.	Alb.
1.	1	25	8	135/85	ST	+	150/90	HT	Sp. FT	OK	120/80	0
2.	1	22	8½	140/110	ST	+	126/76	HT	Sp. FT	OK	N.	0
3.	1	19	8½	145/95	ST	0	145/95	ST	Sp. FT	OK	N.	0
4.	1	19	8¼	162/110	T	+	160/110	T	In. 8½	OK	N.	0
5.	1	28	7	170/120	HT	+	120/90	SPT	Sp. 7¼	Mac.	N.	T
6.	9	46	7	?	?	++	165/95	HT	In. 7¼ Eclamp.	OK	N.	?
7.	1	28	8½	148/100	T	+	160/110	T	In. 8¾	OK	N.	0
8.	1	20	8¾	160/110	HT	+	160/110	VHT	In. FT	OK	N.	0
9.	1	20	8½	154/94	0	++	170/110	T	In. 8¾ P. P. Ec.	OK	N.	0
10.	1	21	8¾	150/104	HT	+	160/105	HT	Sp. FT	OK	N.	0
11.	1	17	8½	160/116	ST	+	160/110	T	Sp. FT	OK	N.	0
12.	1	30	8	130/80	VST	+	150/90	T	In. 8¼	OK	N.	0
13.	1	24	7¼	180/120	HT	++	150/110	HT	In. 7½	OK	N.	0
14.	2	24	8¾	144/90	T	+	130/90	HT	Sp. FT	OK	N.	0
15.	1	26	8¾	148/100	0	+	148/110	SPT	In. FT	OK	N.	0
16.	1	25	8	136/94	0	+	170/90	ST	In. 8¼	OK	N.	0
17.	1	33	8¾	144/100	VST	+	160/100	ST	In. FT	Dead	N.	0
18.	1	19	8¾	120/84	HT	+++	154/100	T	Sp. FT P. P. Ec.	OK	N.	0
19.	1	25	8¾	146/105	SPT	++	140/100	SPT	In. FT	OK	N.	0
20.	1	18	8¾	140/100	ST	+	140/100	ST	Sp. FT	OK	N.	0

granular casts and red blood cells. Her course was as follows:

Date	Intake	Output	Blood Pressure	Albumin	Edema
June 26	0	—	160/90 mm.	ST	0
" 27	600 cc.	800 cc.	140/100 "	SPT	0
" 28	400 "	650 "	138/82 "	SPT	0
" 29	350 "	450 "	120/80 "	SPT	0

The patient was balanced on a fluid intake of 350 cc. and was referred to the toxemia clinic on July 3 with a blood pressure of 120/80 mm. In the clinic, on July 10, the patient had no toxic symptoms. Her blood pressure was 148/96 mm., her urine showed a slightest possible trace of albumin and her fluid intake had averaged 300 to 350 cc. daily, while her output was 50 to 100 cc. greater. On July 24 her blood pressure was 134/86 mm. and the urine showed a slightest possible trace of albumin. On August 15 her blood pressure was 148/92 mm. and the urine as before.

On August 18 the patient was admitted in active labor. The blood pressure was 140/86 mm. and the urine contained a slightest possible trace of albumin. Normal delivery and puerperium ensued and she was discharged in ten days with a negative urine and a blood pressure of 110/70 mm.

This case is an example of one where the treatment was carried on over two months' time and, while complete success was not obtained, nevertheless the patient became no worse. Some may feel that this case should be classed as a low reserve kidney (if there be such a condition), but it seems as though the symptoms developed too early for that.

CASE 5. Mrs. M. N., Nos. 694102 and 697418, white, aged twenty-seven years, 1-para, was first referred to the Boston City Hospital by her local physician on Dec. 24, 1932. Her expected date of confinement was Feb. 4, 1933. Her past history was irrelevant. Since the fifth month of her present pregnancy she had had slight hypertension and albuminuria. On admission she complained of headache and there was marked edema of face and extremities. Her blood pressure was 190/140 mm. The urine showed a heavy trace of albumin with granular casts and red and white blood cells. There was no fixation of gravity and the blood chemistry was within normal limits. The patient was balanced on a fluid intake of 1050 cc. Her blood pressure finally came down to 140/90 mm. and her urine became albumin free. She was discharged back to her family doctor on Jan. 8, 1933 with instructions to follow dehydration therapy. After discharge, the patient developed a cold and did not keep up her fluid balance. As a result she was referred back to the hospital on January 21 by her doctor who said that her blood pressure was 175/110 mm. On admission she progressed as follows:

Date	Intake	Output	Blood Pressure	Albumin	Edema
Jan. 21	0	650 cc.	154/108 mm.	T	++
" 22	500 cc.	1250 "	150/100 "	ST	+
" 23	1200 "	1620 "	150/110 "	SPT	0
" 24	800 "	1700 "	138/90 "	ST	0
" 25	1300 "	1400 "	140/90 "	SPT	0
" 26	1300 "	1300 "	134/90 "	SPT	0
" 27	1150 "	1000 "	130/90 "	T	0
" 28	800 "	950 "	130/90 "	HT	0

In view of patient's nearness to term and increasing albuminuria, labor was induced by castor oil and quinine and by separation of the membranes. Normal labor and delivery of a six pound normal baby ensued in about six hours. The patient made an uneventful convalescence and was discharged home in eleven days with a blood pressure of 110/60 mm. and a slight trace of albumin in the urine.

This case is of interest because of its severity and the fact that she was carried through another whole month after the onset of severe symptoms without obvious damage to either mother or baby. It also demonstrates control of symptoms with dehydration, return of symptoms when dehydration therapy was not carried out and, at the second admission, an almost, but not quite, successful recontrol.

CASE 6. Mrs. M. McA., No. 725599, white, aged thirty-five years, 3-para, was first seen in the toxemia clinic of the Boston City Hospital on June 9, 1933. Her expected date of confinement was Oct. 8, 1933. She had had one full-term instrumental delivery and one four months' miscarriage. Her progress throughout the remainder of her pregnancy was as follows:

Date	Blood Pressure	Albumin	Edema
June 9	160/90 mm.	T	+
July 7	156/98 "	ST	0
July 13	126/84 "	SPT	0
Aug. 14	122/90 "	T	+
Aug. 28	128/94 "	SPT	+
Sept. 11	132/88 "	0	+
Sept. 25	134/92 "	SPT	0
Oct. 2	140/88 "	SPT	0
Oct. 9	144/90 "	0	0

During the above months the patient was carried along with mild dehydration therapy at home. Her fluid balance was kept up with about a 1400 cc. fluid intake. When she was seen on October 9, she complained of frequent headaches and, being at term, was referred into the hospital. She was dehydrated there and her blood pressure came down to 118/70 mm. She started labor spontaneously on October 15 and delivered normally a healthy infant. Her puerperium was uneventful and she was discharged in ten days with a blood pressure of 110/70 mm. and a trace of albumin in the urine.

CASE 7. Mrs. C. A., No. 730975, white, aged sixteen years, 1-para, had been seen several times in the regular out-patient clinic of the Boston City Hospital. Her last catamenia had been on March 23, 1933, and her expected date of confinement was Dec. 30, 1933. Her present pregnancy had been uneventful until December 4, at which time her blood pressure was found to be 168/95 mm. and she showed a slight trace of albumin in the urine, with red and white blood cells and occasional granular casts. She was referred immediately into the hospital. On admission she showed slight edema of the extremities. There was an elevation of the blood nonprotein nitrogen to 40 and the uric acid to 5.7 milligrams per 100 cubic centimeters. Eyegrounds showed slight edema of the retina. Her course was as follows:

Date	Intake	Output	Blood Pressure	Albumin	Edema
Dec. 4	0	700 cc.	168/95 mm.	ST	+
" 5	400 cc.	500 "	140/70 "	ST	0
" 6	500 "	1100 "	140/80 "	HT	0
" 7	700 "	900 "	135/80 "	ST	0
" 8	700 "	1300 "	120/60 "	SPT	0
" 10	600 "	800 "	130/90 "	SPT	0
" 12	700 "	950 "	140/90 "	0	0
" 13	700 "	1100 "	150/90 "	ST	0
" 15	700 "	600 "	140/90 "	SPT	0
" 17	700 "	800 "	135/70 "	SPT	0
" 18	700 "	800 "	120/70 "	0	0

CHART 3

MILD NEPHRITIC TOXEMIAS

Dehydrated Group

Case	Para	Age	Prev. Tox.	Dehydration Started Mo.	B. P.	Alb.	Edema	Finished B. P.	Alb.	Labor	Baby	Discharge B. P.	Alb.
1.	2	28	+	8	154/102	SPT	++	128/90	0	Sp. FT	OK	N.	0
2.	5	43	+	8½	170/102	0	0	125/80	0	In. FT	OK	N.	0
3.	2	30	+	6	140/80	0	0	120/78	0	Caes. FT	OK	N.	SPT
4.	8	37	+	8½	142/100	SPT	++	155/110	ST	Sp. FT	OK	N.	0
5.	5	35	0	8	160/80	SPT	++	140/80	0	Sp. FT	OK	N.	0
6.	2	32	0	8	192/134	SPT	+	180/124	0	In. FT	OK	115/100	0
7.	9	31	+	7	170/92	SPT	0	164/90	0	Sp. FT	OK	N.	0
8.	4	35	+	6	154/104	0	0	142/90	SPT	Sp. FT	OK	148/90	0
9.	4	34	+	5	170/96	0	+	140/90	0	Sp. FT	OK	140/90	0
10.	3	34	+	6	158/90	0	0	120/80	SPT	Sp. FT	OK	120/80	0
11.	4	25	+	8½	162/98	ST	+	120/88	0	Sp. FT	OK	N.	0
12.	3	26	+	5	152/92	SPT	+	120/80	0	Sp. FT	OK	138/100	0
13.	3	31	+	4½	170/82	SPT	++	120/70	0	In. FT	OK	170/82	0
14.	5	24	+	7½	158/98	0	++	164/114	ST	Sp. FT	OK	N.	0
15.	5	33	+	5½	140/90	0	+	120/60	0	Sp. FT	OK	N.	SPT
16.	2	19	+	3½	164/94	0	++	120/80	0	Sp. FT	OK	N.	0
17.	4	27	0	6½	148/88	0	0	140/98	0	Sp. FT	OK	N.	0
18.	2	23	?	4	140/90	SPT	+	124/77	SPT	Sp. FT	OK	N.	0
19.	5	27	+	8	156/102	SPT	+	160/102	SPT	Sp. FT	OK	N.	0
20.	3	27	+	8½	190/100	SPT	+	150/90	SPT	Sp. FT	OK	136/82	0
21.	1	24	0	7½	140/96	T	++	120/80	HT	In. 8½	OK	N.	VST
22.	5	31	0	8¾	150/95	SPT	0	130/80	SPT	Sp. FT	OK	N.	0
23.	2	40	0	8½	150/92	SPT	+	130/90	T	In. 8¾	OK	N.	VST
24.	1	21	0	8½	165/100	SPT	0	150/80	SPT	In. FT	OK	N.	VST
25.	2	28	+	8	140/95	ST	0	130/80	0	Sp. FT	OK	N.	0
26.	1	24	0	8¾	144/98	0	+	130/80	0	Sp. FT	OK	N.	0

CHART 4

MILD NEPHRITIC TOXEMIAS

Nondehydrated Group

Case	Para	Age	Prev. Tox.	Treatment Started				Finished				Discharge	
				Duration of Preg. Mo.	B. P.	Alb.	Edema	B. P.	Alb.	Labor	Baby	B. P.	Alb.
1.	2	22	+	7	144	0	0	170	0	In. FT	OK	N.	0
2.	5	41	++	8½	90/156	T	+	110/160	T	Sp. 8¾	OK	N.	?
3.	1	23	0	8¼	100/145	SPT	0	90/130	SPT	In. FT	OK	N.	0
4.	1	24	0	8	90/160	SPT	+	80/175	0	Sp. FT	OK	N.	0
5.	6	27	+	8¼	110/170	SPT	+	105/130	0	Sp. FT	Mac.	N.	?
6.	1	26	0	7½	110/145	SPT	0	80/122	SPT	Sp. 8	Mac.	?	?
7.	4	31	++	7¾	110/164	SPT	+	90/?	T	In. 8	OK	?	?
8.	12	39	++	7½	114/160	SPT	++	180	T	Sp. FT	OK	N.	0
9.	1	24	0	7	110/138	SPT	0	118/170	SPT	Sp. FT	OK	N.	0
10.	1	27	0	7½	90/144	SPT	+	110/160	T	Sp. 8½	OK	N.	0
11.	10	41	+	6	102/140	0	+	100/128	HT	In. 7¼ Pre. Sep.	Mac. of Plac.	N.	0
12.	8	44	++	8½	100/140	SPT	+	100/144	ST	Sp. FT	OK	N.	0
13.	5	36	0	8	94/165	0	+	80/150	SPT	Sp. FT	OK	N.	0
14.	4	35	+	8½	100/140	0	++	94/130	SPT	Sp. FT	Dead	N.	0
15.	2	25	+	8¾	100/140	T	+	90/140	T	Sp. FT	OK	N.	0
16.	1	24	0	8¼	95/148	ST	+	100/150	SPT	Sp. FT	OK	N.	0
17.	1	21	0	7	80/145	SPT	0	75/130	SPT	Sp. FT	OK	N.	0
18.	4	32	++	8	90/140	0	0	60/150	ST	In. 8½	OK	N.	0
19.	2	34	+	8½	95/190	ST	+	105/150	SPT	In. 8¾	OK	N.	0
20.	1	28	0	8¾	100/130	ST	+	100/150	0	In. FT	Mac.	N.	0
21.	1	21	0	7½	80/140	T	+	100/135	T	In. 8	OK	N.	0
22.	1	25	0	7½	100/138	SPT	0	90/130	0	Sp. 8	OK	N.	0
23.	1	21	0	8	90/170	ST	+	90/145	T	In. FT	OK	N.	SPT
24.	1	21	0	8¾	110/130	ST	+	95/140	ST	Sp. FT	OK	N.	SPT
25.	7	38	+	8	80/144	0	0	90/180	SPT	Sp. FT	OK	N.	0
26.	12	39	+	8¾	86/120	ST	+	112/155	ST	Sp. FT	OK	N.	0
					80			95					

Vol. 193

She was balanced on an intake of 700 cc. of fluid daily and referred back to the out-patient clinic. Her blood pressure and urine albumin remained within normal limits, and, on December 26, she was admitted in active labor and delivered. Her admission blood pressure was 130/100 mm. and there was a slight trace of albumin in the urine. Sediment showed no blood cells, but occasional granular casts. Ten days postpartum her urine showed no albumin, but occasional granular casts and her blood pressure was 94/60 mm.

This case is interesting because, although she never showed a great deal of external edema, her output remained for two weeks considerably above her limited intake, showing a great deal of stored fluid. In addition, her normal blood pressure (94/60 mm.) was low, which makes the moderate elevation she had relatively greater.

b. Mild nephritic toxemias.

Charts 3 and 4 represent the groups of the mild nephritic toxemias. There were twenty-six cases in each of these groups. All but two of the dehydrated group went to full term and nine in the control group terminated prematurely. There were no macerated fetuses in the dehydrated group but there were four macerated fetuses and one stillbirth in the control group. One case in the control group developed *abruptio placentae*.

CASE REPORTS

CASE 1. Mrs. C. S., No. 724985, white, aged twenty-eight years, 2-para, was admitted to the Boston City Hospital on Oct. 10, 1933. Her expected date of confinement was Nov. 4, 1933. She had had scarlet fever in childhood. Her last pregnancy went to full term, but she had had hypertension and albuminuria the last two months. The present pregnancy had been uneventful except for frequent headaches and edema of the hands and feet since the second month. She was first seen in the out-patient department on July 12, at which time she had a blood pressure of 130/82 mm. and a slightest possible trace of albumin in her urine. She was cared for in the regular prenatal clinic until October 9, at which time her blood pressure was found to be 154/102 mm. Her urine showed a slight trace of albumin with red and white blood cells and occasional granular casts. She showed moderate edema of the face, hands and feet. She was referred into the hospital and her course there was as follows:

Date	Intake	Output	Blood Pressure	Albumin	Edema
Oct. 10	0	1200 cc.	150/100 mm.	ST	+++
" 11	1200 cc.	1100 "	140/75 "	SPT	+
" 12	1000 "	1300 "	125/70 "	—	0
" 13	1300 "	1500 "	110/70 "	0	0
" 14	1500 "	1900 "	115/70 "	0	0
" 15	1600 "	1100 "	110/70 "	—	0

On October 16 the patient was discharged back to the toxemia clinic with a blood pressure of 128/90 mm. and with no albumin. While in the hospital the phthalein test was 55 per cent. Examination of eyegrounds revealed numerous small areas of exudate with hemorrhage. Her fluids were balanced at an intake of about 1000 cc. daily. On October 23 the pressure was 136/90 mm. and there was the slightest possible trace of albumin. On November 5 the patient was admitted to the hospital in active labor. Her blood pressure was 136/70 mm. and the urine was negative for albumin. She was delivered normally of a healthy infant and had an uneventful puerperium. Her discharge blood pressure was 120/80 mm. and the urine was negative.

CASE 2. Mrs. N. P., No. 725,717, white, aged forty-three years, 5-para, was referred into the Boston City Hospital from the prenatal clinic on Oct. 5, 1933. The expected date of confinement was Oct. 27, 1933. She had had four normal full-term pregnancies and hypertension during the last five months of the last pregnancy. She had first come to the clinic on July 17 and at that time her blood pressure was 154/90 mm. This moderate hypertension had persisted and, on October 4, had increased to 170/102 mm. At this time she was referred into the hospital for dehydration therapy. On admission the urine showed a slightest possible trace of albumin with occasional hyalin casts and red blood cells. The patient's eyegrounds and blood chemistry were negative. Under treatment the blood pressure came down to 125/80 mm. and albumin became negative. She was balanced on 700 cc. daily intake and was referred back to the toxemia clinic on October 12. Four days later she came to clinic and admitted not having followed instructions. Her blood pressure had risen to 166/100 mm. She was readmitted to the hospital and labor was induced with castor oil and quinine. Her convalescence was normal and she was discharged with normal blood pressure and negative urine.

c. Severe nephritis.

Charts 5 and 6 represent the dehydrated and control groups of severe nephritic toxemias. There are nineteen cases in each of these groups. Naturally our worst results were obtained in this group but even so the dehydrated cases did considerably better than those not dehydrated. Five of the cases, which, under dehydration were carried to full term, had been induced in their previous pregnancies at periods varying from six to eight months.

CASE REPORTS

CASE 1. Mrs. B. M., O.P.D. No. 307061, white, aged thirty-three years, 1-para, was first seen in the regular prenatal clinic of the Boston City Hospital on May 22, 1933. Her expected date of confinement was July 13, 1933. Her past history was negative except for scarlet fever in childhood. When first seen her blood pressure was 148/94 mm. and the urine was negative. On June 28 the pressure was 165/120 mm. and there was a slight trace of albumin in the urine. The patient was referred to the toxemia clinic, and there on July 3 the blood pressure was 190/140 mm. and the urine showed a trace of albumin with red and white blood cells, but no casts. The patient complained of severe headaches and moderate edema of the face and extremities. She was immediately referred into the hospital. On admission the patient was given 100 cc. of 50 per cent glucose intravenously. Blood chemistry and eyegrounds were negative. Her progress in the hospital follows:

CHART 5

SEVERE NEPHRITIC TOXEMIAS

Dehydrated Group

Case	Para	Age	Prev. Tox.	Dehydration Started Mo.	B. P.	Alb.	Edema	Finished B. P.	Alb.	Labor	Baby	Discharge B. P.	Alb.
1.	1	33	0	8½	190/140	T	+++	135/95	HT	Sp. FT	OK	140/100	SPT
2.	2	23	+	7½	140/70	HT	0	128/80	ST	Sp. 8	OK	N.	0
3.	3	27	+	6	160/120	T	+++	170/130	HT	Sp. 6½	Mac.	N.	ST
4.	2	22	+	2	142/84	T	+++	120/80	HT	In. 8 Caes.	OK	N.	ST
5.	5	31	+	6½	162/110	SPT	+++	130/80	SPT	In. 8½	OK	140/100	SPT
6.	2	29	+	6	170/110	T	+	130/80	SPT	In. 7	Mac.	N.	0
7.	2	26	+	8½	140/90	T	++	135/90	HT	In. FT	OK	N.	ST
8.	3	28	+	6¼	152/112	0	+	166/112	T	In. 8½	OK	N.	SPT
9.	16	36	+	7½	170/110	HT	+	170/110	HT	In. 8	OK	160/110	T
10.	8	31	+	6	222/128	0	+	160/110	0	Sp. 8½	OK	170/110	0
11.	2	22	+	8	196/98	SPT	+	170/100	0	Sp. FT	OK	N.	0
12.	10	39	+	6	204/110	HT	+++	158/104	HT	In. 6½	Mac.	140/100	ST
13.	6	34	+	7½	162/94	HT	++	138/90	HT	In. 8½	OK	N.	SPT
14.	3	27	+	6	224/140	T	+++	170/130	SPT	Sp. 7	Mac.	160/110	0
15.	10	46	+	8	155/90	HT	++	140/80	ST	Caes. FT	OK	N.	SPT
16.	7	32	+	7½	160/100	SPT	++	135/85	0	Sp. FT	OK	N.	0
17.	2	29	+	7	150/100	HT	+	114/74	ST	In. 7½	OK	N.	SPT
18.	9	40	+	8¼	198/118	T	++	160/110	T	In. 8½	OK	150/80	SPT
19.	1	23	0	6¼	190/90	T	++	130/80	SPT	Sp. 7	Mac.	N.	0

CHART 6

SEVERE NEPHRITIC TOXEMIAS

Nondehydrated Group

Case	Para	Age	Prev. Tox.	Treatment Started Mo.	B. P.	Alb.	Edema	Finished B. P.	Alb.	Labor	Baby	Discharge B. P.	Alb.
1.	3	34	+	7	178/100	ST	++	180/100	T	Sp. 7½	Mac.	150/90	SPT
2.	9	40	+	7	178/110	HT	+	140/90	HT	In. 7	OK	120/80	0
3.	3	24	+	8	175/120	SPT	+	190/140	HT	In. 8½	OK	N.	0
4.	11	38	+	7½	222/142	SPT	++	240/150	HT	In. 8	Mac.	Died	
5.	5	35	+	6¼	198/100	T	++	160/110	HT	In. 7	Mac.	120/80	ST
6.	1	40	0	8¼	160/100	HT	+	214/110	HT	In. 8½	OK	130/90	?
7.	7	45	+	8½	160/100	T	+	150/90	HT	Sp. FT	OK	140/90	?
8.	9	37	+	7½	160/95	ST	+	160/100	?	Sp. 8	Mac.	140/80	0
9.	6	28	+	7½	164/92	T	++	140/90	T	Sp. 8	OK	N.	0
10.	5	37	+	8¼	172/120	T	+	160/100	T	In. FT	Mac.	N.	0
11.	2	34	+	5	150/100	HT	+++	134/90	T	In. 6	Mac.	N.	VST
12.	1	31	0	7¾	180/110	ST	++	200/110	ST	In. 8	OK	N.	0
13.	2	32	0	6¾	134/100	HT	+	120/80	VST	In. 7¼ Caes. Pre.	Mac. Sep. Plac.	N.	SPT
14.	8	37	+	6	130/90	ST	+	160/90	ST	In. 6	Mac.	N.	SPT
15.	6	33	+	7½	138/90	T	+	148/104	T	In. 7¾	OK	N.	ST
16.	7	30	+	6½	134/110	0	++	136/98	SPT	Sp. 7	Mac.	N.	0
17.	3	38	+	3	170/120	0	+	220/140	T	In. 6	Mac.	162/130	0
18.	7	43	+	6	160/100	HT	++	180/110	HT	In. 6¾	OK	140/100	ST
19.	7	?	++	8	160/100	T	+	160/110	ST	Sp. FT	OK	N.	SPT

Date	Intake	Output	Blood Pressure	Albumin	Edema
July 3	0	—	190/140 mm.	T	+++
" 4	700 cc.	1700 cc.	160/105 "	HT	+
" 5	750 "	900 "	155/105 "	HT	+
" 6	500 "	300 "	150/110 "	HT	0
" 7	300 "	450 "	150/100 "	—	0
" 9	250 "	750 "	150/94 "	T	0
" 12	500 "	600 "	145/100 "	HT	0
" 17	600 "	700 "	135/95 "	HT	0

The patient started up in spontaneous labor and was delivered of a normal baby by low forceps on July 17. The convalescence was uneventful and the patient was discharged home with a pressure of 140/100 mm. and a slightest possible trace of albumin in the urine.

CASE 2. Mrs. R. G., No. 697743, white, aged twenty-three years, 2-para, was referred into the Boston City Hospital from the toxemia clinic on Jan. 24, 1933. Her past history was negative. Her last pregnancy had been terminated at eight months for toxemia. She had been followed in the toxemia clinic since August and her pregnancy had been uneventful until January, at which time she began to show evidences of hypertension and albuminuria. On January 24 she had a blood pressure of 140/120 mm. and a heavy trace of albumin in the urine, with red and white blood cells, but no casts. She was referred to the hospital. Eyegrounds were negative, there was no fixation of gravity and the phthalein test was 50 per cent. She had no toxic symptoms and no edema. Under drastic dehydration therapy (averaging around 250 cc. daily intake), her blood pressure came down to 110/70 mm. and her urine became negative for albumin within a week. At the end of this time she started in labor spontaneously and delivered a normal baby. Although she was only eight months along by dates, the baby gave no evidence of prematurity. The patient had a normal puerperium and was discharged on February 11 with a blood pressure of 110/70 mm. and a negative urine.

CASE 3. Mrs. E. B., No. 725,725, white, aged twenty-seven years, 3-para, was admitted to the Boston City Hospital on Oct. 16, 1933. She gave a history in her last two pregnancies of two macerated fetuses occurring at seven and eight months. Her present pregnancy had been symptomless, but on admission she had a blood pressure of 224/140 mm. Her blood pressure had been high on two previous occasions in the O.P.D. Her urine showed a trace of albumin with hyalin and granular casts and red and white blood cells. The blood chemistry was within normal limits. Basal metabolism was minus one. Eyegrounds were negative. There was a high fixation of gravity between 1.021 and 1.030. The patient was placed on a dehydration régime and her fluid intake was balanced between 500 and 700 cc. daily. At the end of the first week of treatment the blood pressure was 190/140 mm. and there was still a trace of albumin in the urine. At the end of the second week the blood pressure varied between 150/120 mm. and 170/130 mm. and the albumin had decreased to a slight trace. In another week she became practically albumin-free. From then until November 23 her pressure persisted around 170/120 mm. and the albumin varied between none and the slightest possible trace. On November 22 the fetal heart could not be heard, in spite of the fact that the patient had had no symptoms and had been albumin-free for several days preceding. On November 23 the patient went into labor spontaneously and delivered a macerated fetus. Her convalescence was uneventful and she was discharged home in two weeks with a blood pressure of 160/110 mm. and a slight trace of albumin in the urine.

CASE 4. Mrs. E. M., No. 701983, white, aged twenty-two years, 2-para, was first seen in the toxemia clinic of the Boston City Hospital at the second month of her second pregnancy. Her first pregnancy had terminated at six months by maceration of the fetus and, later, separation of the placenta. Six weeks after this she had had a blood pressure of 140/95 mm. and a trace of albumin in the urine. Her present expected date of confinement was Apr. 27, 1933. When first seen Oct. 31, 1932, her blood pressure was 120/78 mm. and her urine showed a trace of albumin.

In November her pressure varied between 124/82 mm. and 142/84 mm. and her urine albumin varied between none and a slightest possible trace. In December her pressure was 134/88 mm. and the albumin had increased to a trace with red and white blood cells, but no casts. She had no toxic symptoms but was sent into the hospital on December 28 for dehydration therapy. Her fluid intake was balanced at about 1100 cc. daily. The blood chemistry, eyegrounds and gravity fixation tests were negative. The patient had a red blood cell count of 3,500,000 and a hemoglobin of 75 per cent. Her phthalein test was 40 per cent. On January 10 she was discharged back to the toxemia clinic with a blood pressure of 110/64 mm. and a slightest possible trace of albumin.

During the rest of January her blood pressure varied between 138/90 mm. and 150/100 mm. and her urine albumin between a slight trace and a trace. Hyalin and granular casts and red blood cells appeared for the first time in her urine. The patient admitted being careless at times about her fluid intake and noticed that when she did not restrict fluids she became edematous and felt poorly. Her intake was decreased from 1100 cc. to 750 cc. daily. During February her blood pressure varied between 126/94 mm. and 144/92 mm. and her albumin from a trace to a heavy trace. She had slight edema and her output was not exceeding her intake. On March 6 the patient's blood pressure was 162/112 mm. and she had a slight trace of albumin. Because of the patient's extreme desire for a child, the pregnancy was allowed to continue and she was referred back to the hospital.

On admission this time her blood chemistry was again within normal limits. Her hemoglobin had dropped to 50 per cent. There was still no gravity fixation, but her phthalein test had decreased to 20 per cent. During the next two weeks her blood pressure came down promptly to 120/80 mm., but her albumin varied between a trace and a heavy trace. Her intake during this time had been decreased to between 200 and 400 cc. daily. On March 18 the patient complained of the dehydration and was allowed more fluids. She promptly became edematous and complained of malaise, dizziness and headache. On March 27, because of the persistence of a trace to heavy trace of albumin and because the patient was then one month from term and the baby seemed of fair size, interruption was advised. She was delivered of a five-pound healthy baby by cesarean section under spinal anesthesia. She had an uneventful convalescence and was discharged home with her baby on April 13. Her discharge blood pressure was 120/80 mm. and the urine showed a trace of albumin. Six weeks later her blood pressure was 130/98 mm. and the urine showed a slight trace of albumin with rare red blood cells and granular casts in the sediment.

Just how much damage was done to this woman's kidneys by allowing her to go through this pregnancy cannot be proved, but her extreme desire for a child left no choice. At any rate, an excellent opportunity was given to use dehydration over a long period of time on a case which was considered to be that of a severe chronic nephritic, in spite of the lack of great hypertension.

CASE 5. Mrs. A. S., No. A8744, white, aged thirty-one years, 5-para, was first seen in the O.P.D. of the Newton Hospital on July 27, 1933. Her blood pressure at this time was 162/100 mm. and her urine showed a slightest possible trace of albumin with hyalin casts. She was referred to the hospital. On admission she gave an history of four previous full-term normal deliveries with hypertension in at least the last two. She showed moderate edema of the extremities, but otherwise had no toxic symptoms. Blood chemistry and renal function tests were normal. The patient was placed on dehydration therapy and fluids were balanced at between 750 and 900 cc. daily. She was very uncooperative and had to be watched closely to prevent extra fluid ingestion. Her blood pressure came down gradually to 130/80 mm. over a period of twelve days and she was referred back to the outpatient clinic. She did not follow instruction at home, and on September 14 her blood pressure had risen to 228/142 mm. and her urine showed a trace of albumin. She was, at this time, eight and a half months pregnant and it seemed inadvisable to delay delivery further. Hence, her membranes were ruptured, and she had an uneventful labor, delivery and convalescence. She was discharged in two weeks with a negative urine and a blood pressure of 140/110 mm.

Chart 7 represents the summarized statistics of all groups. In the dehydrated group, thirteen cases had to be induced prematurely as compared with twenty-four cases (almost twice as many) in the control group. The blood pressure was decreased in 59 cases and the albumin in 26 cases of the dehydrated group as compared with 24 cases and 12 cases in the control group. Five cases of maceration of the fetus occurred under dehydration while in the control group there were fifteen cases. Furthermore all the cases of maceration in the dehydration series occurred in the severe type of nephrities whereas five of the control cases occurred in the preeclamptic and mild nephritic groups. No cases of eclampsia or *abruptio placentae* occurred under dehydration but two cases of *abruptio*, one case of antepartum eclampsia and two of postpartum eclampsia occurred in the control group. In the cases showing albumin or persistent hypertension on discharge, the control group with 15 seems definitely better than the dehydrated group with 29. This superiority is probably more apparent than real because, in seven of the control group, discharge findings were not recorded and it is possible that other control records were not accurately kept, especially as far as taking a discharge blood pressure or a catheterized specimen of urine was concerned.

The results of these studies make it appear that dehydration has a very definite place in the treatment of the pregnancy toxemias. The series is small but the results were very satisfactory and it is hoped that similar studies will be made and reported by other obstetricians.

CHART 7

SUMMARIZED STATISTICS ALL GROUPS

	Pre-eclamptic		Mild Nephritic		Severe Nephritic		All Groups	
	De-hyd.	Con-trol	De-hyd.	Con-trol	De-hyd.	Con-trol	De-hyd.	Con-trol
Total number cases	20	20	26	26	19	19	65	65
Primiparae	11	18	3	13	2	2	16	33
Multiparae	9	2	23	13	17	17	49	32
Cases carried to full term	16	12	24	17	5	3	45	32
Cases induced prematurely	3	7	2	5	8	12	13	24
B. P. decreased in	20	6	23	11	16	7	59	24
B. P. unchanged in	0	6	0	2	1	2	1	10
B. P. increased in	0	8	3	13	2	10	5	31
Albumin decreased in	10	2	8	7	8	3	26	12
Albumin unchanged in	9	8	12	10	6	9	27	27
Albumin increased in	1	10	6	9	5	7	12	26
Cases discharged with alb. or inc. b. p.	5	1	10	2	14	12	29	15
Number macerated fetuses	0	1	0	4	5	10	5	15
Number cases developing eclampsia	0	3	0	0	0	0	0	3
Number of cases of *Abruptio Placentae*	0	0	0	1	0	1	0	2
Maternal mortality	0	0	0	0	0	1	0	1

REFERENCES

1. Arnold, J. O., and Fay, T.: Eclampsia: Its prevention and control by means of fluid limitation and dehydration. Surg. Gynec. Obst. **55**:129 (Aug.) 1932.
2. Elsberg, C. A., and Pike, F. H.: Studies on epilepsy; influence of a general increase or diminution of intracranial pressure upon the susceptibility of animals to convulsive seizures. Am. J. Physiol. **76**:593 (May) 1926.
3. Gamble, J. L., Ross, G. S., and Tisdall, F. F.: The metabolism of fixed base during fasting. J. Biol. Chem. **57**:633 (Oct.) 1923.
4. Gamble, J. L.: Dehydration. New Eng. J. Med. **201**:909 (Nov. 7) 1929.
5. Lennox, W. G., and Cobb, S.: Epilepsy. Medicine **7**:105 (May) 1928.
6. Rowntree, L. G.: The water balance of the body. Physiol. Rev. **2**:116 (Jan.) 1922.
7. Idem: The effects on mammals of the administration of excessive quantities of water. J. Pharmacol. & Exper. Therap. **29**:135, 1926.
8. Thomas, W. H.: Certain physiologic problems arising from forcing of fluids. Penn. M. J. **34**:329 (Feb.) 1931.
9. Irving, F. C.: The vascular aspect of eclampsia. Am. J. Obst. & Gynec. **31**:466 (March) 1936.
10. Smith, G. V. S. and O. W.: Evidence for the placental origin of the excessive prolan of late pregnancy toxemia and eclampsia. Surg. Gynec. Obst. **61**:175 (Aug.) 1935.
11. Idem: Further quantitative determinations of prolan and estrin in pregnancy with especial reference to late toxemia and eclampsia. Surg. Gynec. Obst. **61**:27 (July) 1935.

THE PROBLEM OF EYE INJURIES*

BY CONSTANCE G. HARTWELL, M.D., † AND WILLIAM D. ROWLAND, M.D.†

THE first physician to care for any seriously injured eye has a real responsibility. While the same statement may be made concerning similar injuries to many other parts or organs, it is especially true of the eye because of its delicacy of structure and its importance as a functioning organ. The author will attempt to recall in this paper some of the more serious injuries and discuss their care. Illustrative cases will be used to make these points more practicable.

NATURE OF WOUNDS AND AGENTS

In automobile accidents, although less frequent since the use of nonshatterable glass, lacerated lids are often encountered, while torn wounds may be made by blunt objects exerting considerable force—such as face trauma, when thrown against the steering wheel or instrument board. This is equally true of accidents in industry, among playing children, in certain sports and in fistic battles.

Chemical burns to the lids, conjunctiva and cornea, because of rapid infiltration and rapid necrosis are very destructive if the solutions are strong or not neutralized very quickly. Hot liquids or flames are quite similarly destructive or distort by scar contractions.

Contusions to the globe made by blunt objects which strike the eye (such as tennis- or snowballs) or against which the eye is forced (doorknobs or articles of furniture) may result in a torn iris, dislocated lens, intraocular hemorrhage and—not infrequently—torn or detached retina.

Penetrating and perforating wounds may be even more dangerous. A perforating wound is one producing a hole large enough for the escape of aqueous or vitreous humor; a penetrating wound is one piercing the globe and usually injuring deeper structures and in which there may be little or no leaking of fluids. Wounds through the cornea usually injure the iris and lens and result in a traumatic cataract, while those through the thin overlying conjunctiva and tough sclera tap the vitreous and injure the choroid and retina. In wounds over the area surrounding the cornea, the underlying ciliary body is usually cut or torn and creates a situation quite as serious as any joint wound, for this is the danger zone because of the greater possibility of sympathetic trouble in the fellow eye.

Agents producing these wounds are of a wide variety. They may be small flying particles arising from industrial or domestic pursuits which remain within the globe or they may be larger objects such as nails, pens, sticks, sharp tools, scissors, broken glass and so forth. Injuries from arrows and B.B. shots from air guns among playing children are too frequent and the sale of these toys should be prevented by law. Explosions may carry stone, earth, and metal fragments most of which carry infection which has an immediate effect. It is, however, astonishing how many times the eye remains clean, either because the agent was sterile on entering (such as chips from a steel tool, doubtless sterilized by friction heating) or because the eye tissues are unusually resistant to ordinary pathologic organisms. The micro-organisms which are found most frequently are pneumococci, staphylococci and the streptococci. Others are found, of course, and one is usually concerned about tetanus.

Chemically contaminated agents or chemically resolving foreign bodies add further problems. Iron and steel usually produce siderosis by dissolving, the fine particles scattering through the intraocular structures and setting up toxic inflammation. Copper acts similarly. Occasionally, foreign bodies are encysted, usually the inert ones, and may be retained with little danger.

MANAGEMENT AND THERAPEUTICS

In all accidents a very careful history from the patient or any witness is frequently most informative and helpful. In industrial cases, early notification of the insurer is advisable. In most cases, consultation is likewise advisable—even among mature ophthalmologists—for the benefit of the patient, first, and for the support of the attending physician.

The first surgical repair of torn or lacerated lids—if carefully done with exacting tissue approximation, using fine sutures—will insure good function and, likewise, good cosmetic results in most cases. Otherwise, much difficulty lies ahead for the one who must do the necessary plastic repair.

Chemical burns should have immediate neutralization, if possible, and the application of sterile lubricants to prevent adhesions. Occasionally, early plastic operations are advisable. The cornea is particularly vulnerable and any extensive burn will doubtless result in blindness through corneal opacity or possible intraocular infection.

*Presented before the Staff, Massachusetts Memorial Hospitals, November 29, 1935.

From the Massachusetts Memorial Hospitals and the Boston University School of Medicine, Boston, Eye Section.

†Hartwell, Constance G.—Interne at the Massachusetts Memorial Hospitals, Boston. Rowland, William D.—Professor of Ophthalmology, Boston University School of Medicine. For records and addresses of authors see "This Week's Issue," page 303.

Nonpenetrating wounds should have early and careful management—usually cold applications and quiet until any bleeding stops, almost always the use of atropine to keep the eye at rest, absorptive treatment to clear hemorrhage and exudates and, if there is a detached retina, more quiet and continued rest in bed. If taken early, many badly traumatized eyes may be restored to useful or full function, but a delay of only a day or two may render the salvaging much more difficult, if not wholly impossible.

Penetrating or perforating wounds to the globe present two main problems. First, the possibility of infection; secondly, the salvaging of the eye. The safest rule to follow is that of having every injured eye properly investigated, no matter how trivial the injury. Many people suffer penetrating foreign bodies without being conscious of an accident.

The first care, usually given by the family or industrial physician, should be a careful cleansing with sterile saline (almost always available), the instillation of a noncorrosive antiseptic and atropine (if available) and the application of a sterile pad best held in place by adhesive. The next step is the help of an ophthalmologist who may try a hand magnet to detect a foreign body and place the patient in a hospital where x-ray detection and localization of possible foreign bodies may be made. Plans for such surgical repair or removal of foreign bodies as may be found necessary should be carried out without delay. A giant magnet is often necessary, but not all hospitals have this important apparatus. Antitetanic serum should be given and with awareness of possible serum reaction.

Penetrating wounds usually close themselves. Perforating wounds should be closed by sutures, if in the sclera, or covered by conjunctival flaps, if in the cornea or at the limbus. Virulent infections are usually apparent within a very short time and almost always defeat any effort at control, while moderate or delayed infections may often be controlled. Therapeutics, after the initial antitetanic serum, should consist of large doses of salicylates or foreign proteins. Boiled whole milk in 10 cc. intramuscular injections, typhoid vaccines more efficiently given intravenously and diphtheria antitoxin subcutaneously are the usual foreign protein agents resorted to. These are repeated daily or at longer intervals for such periods as are necessary in a given case. Serum reactions must be watched and controlled. Atropine is almost always necessary to keep the eye at rest, intraocularly, by splinting the ciliary muscle and dilating the pupil, always with alertness for a rise of intraocular pressure.

X-ray may not be necessary in selected cases but many intraocular foreign bodies are wholly overlooked when there is no excuse for it. A few substances lodging within the eye may not be x-ray visible. One can make no mistake in having radiographs made—it is much better to err on the safe side.

Nonmagnetable bodies, such as wood, glass, lead, stone and so forth, are more difficult of removal especially if in the vitreous chamber. If small and probably clean, it is sometimes better judgment to allow them to remain, providing the eye is not too greatly traumatized and becomes quiet within a reasonable time. Larger masses of these materials usually injure so badly that enucleation becomes advisable early.

Sympathetic involvement of the other eye is possible within a few weeks or may be delayed many years—as long as forty years have been reported in authentic cases. This inflammation may show first in the irritative stage simply as tearing and photophobia or, perhaps, as reduced vision, if developing in the choroid in the fundus. A few eyes have been saved by heroic therapy when this serious condition has ensued, but enucleation of the injured eye is usually advised. The best judgment of capable men is often taxed in such emergencies. An injured eye, from which a foreign body has been removed or in which one still resides, should always be kept under observation and the patient cautioned accordingly. During the World War relatively few cases of sympathetic ophthalmitis were encountered, possibly because of early care and the use of antitetanic serum routinely. In children, we meet this condition less frequently, but, when it does develop, it is much more difficult to control.

CASE REPORTS

CASE 1. Torn lid.

C. B., aged six, while chasing a cat in his back yard, September 8, 1932, fell against a drain at the corner of his home and tore the lower right lid on a projecting spike. Early care was given by the family physician and the patient was sent to the hospital. The following day under general anesthesia further examination and surgical repair were carried out. A ragged tear extended from the lower punctum outward and down one inch, including the skin, muscle, and conjunctiva. The globe fortunately was not injured.

The conjunctiva was first closed by interrupted sutures, then by deep sutures the muscle was carefully brought into exact position at the inner canthus with a smooth lid margin and the skin exactly approximated by interrupted sutures. Tetanus antitoxin, 800 units, was given intragluteally. The eye was covered by sterile dressing.

He left the hospital on the third day and dressings were continued at the office. The wound healed by first intention.

End-Result:

In one month the lid was in good condition and had good function, almost no scarring and a draining lower canaliculus.

CASE 2. Chemical burn.

M. F., aged fifty-five, while applying labels to packages for delivery from a large department store February 6, 1936, splashed *water glass* (used for its

quick drying effect) into the left eye, producing a second degree burn to the lid and conjunctiva of the lower culdesac. First aid, which consisted of washing with water and instilling oil, was applied by the local medical service. When seen two hours later the above-mentioned burn was found, but the cornea was not injured, probably saved by the instantaneous involuntary closing and rotating of the eye upward.

Holocain, 1 per cent, in sterile ointment was used frequently and the lids kept quiet by sterile dressing. Water glass is salicylate of sodium or potassium and has an alkaline strength about equal to washing soda. Diluted vinegar would have been a good neutralizer for use by those giving first aid.

End-Result:

In eleven days the eye was white and the slit lamp showed the cornea clear.

CASE 3. Contusion to the globe by a snowball.

R. H., aged twelve, was hit in the right eye by a playmate throwing snowballs on January 12, 1932. Home care was carried out and four days later the family physician was called because of a painful and almost blind eye.

When examined on the fifth day, the lids were swollen and closed, the eye was moderately red, there was much photophobia and lacrimation, hemorrhage obscured the iris, vision was reduced to hand motion and the fundus was not seen.

The eye was kept quiet by judicious use of atropine and periodic hot compresses and absorption was aided by dionin. As the eye cleared, it was apparent that bleeding had extended into the anterior vitreous body and this was slow in clearing. No detachment of the retina had occurred; at any rate none was found when the eye was clear enough to study that structure adequately with the ophthalmoscope at the end of six months. The anterior vitreous remained cloudy and was finally cleared by potassium iodide.

This case shows the folly of delayed first care and the good results to be obtained by persistent treatment.

End-Result:

In one year the eye was clear and normal vision was obtained by a correction of myopia and myopic astigmia, slightly greater in amount than required by the fellow eye.

CASE 4. Air rifle shot into the eye.

L. L., aged eleven, while playing near his home in a fine suburban residential neighborhood, was shot in the right eye by another boy carrying an air rifle in March, 1931, early evening. Cold applications and rest were carried out. The next morning the eye showed redness and swelling over the lower outer quadrant and a large, irregular, and poorly reacting pupil with vision reduced to 20/100 from 20/30 (previously recorded). No apparent lesion was discerned by the ophthalmoscope. After anesthetizing with butyn, a search found a B.B. shot under the conjunctiva which was buried in the lower margin of the external rectus muscle. This was removed as an office procedure with some difficulty.

The eye was kept quiet with atropine and covered with a sterile pad for a few days. Within two weeks pigmentation and retinal distortion became evident just below and in front of the macula, being a reaction in the choroid and retina from the trauma to the globe. Apparently the shot struck a glancing blow almost tangential to the eyeball. Otherwise, at the distance of twenty feet, the shot would have doubtless penetrated into the vitreous.

End-Result:

The eye rapidly became white, but the retinal lesion was active for more than six months. With a correction for mixed astigmia, the vision is 20/30 and Jaeger No. 1 with difficulty. Two small paracentral scotomata (blind spots) still remain, as of December 28, 1933.

CASE 5. Perforating wound over the limbus by a large splinter of glass; prolapse of iris and vitreous.

M., aged eighteen months, while watching his mother working at a kitchen cabinet, was injured when a package fell from a top shelf striking and breaking a conical glass orange squeezer, a splinter from which apparently fell into his right eye. The family physician gave ether and made sufficient examination to determine that a sizable hole had been cut into the globe. After telephone consultation, mercurochrome and atropine were instilled, a sterile pad was applied and the child was sent to the hospital. After re-examination of the eye was made, tetanus antitoxin (1000 units) was given intramuscularly and plans made for operation the next day.

January 21, 1936, again under anesthesia, more careful examination was possible and an irregular hole, 4 mm. in greatest width and filled with prolapsed iris and vitreous, was found over the limbus at seven o'clock. It was clinically clean. The lens was clear (?) and in normal position. The fundus was not studied with an ophthalmoscope. No fragments of glass were found then or by the family physician the previous evening. Presumably one rather large splinter had—largely by its weight—produced the perforation. The prolapsed iris and extruding vitreous were excised and a very large conjunctival flap was brought from below and sutured above the cornea, entirely covering the hole as well as the cornea. Although the eye was still clinically clean, a very guarded prognosis was given for saving the injured eye, for safety to the fellow eye (because of the ciliary body injury) or for useful vision.

At the second dressing on the sixth day, the conjunctiva had returned to its original position except for covering the perforation and extending over the cornea about 3 mm. at that point. The eye was left open after the twelfth day, being white and clean with a good fundic reflex and, apparently, good vision.

End-Result:

On March 16, 1936, the eye was white and clean. The lens showed cataractous change in the region of perforation.

CASE 6. Multiple extensive lacerations of the eyeball and lids by glass from a broken eye glass.

Mrs. A., aged fifty, while playing golf, July 21, 1932, suffered a severe injury to her right eye when struck by a ball hit by a mashie at forty yards. After care by her physician both for the eye and the shock incident to the severe blow, she was seen the next morning at the office. Multiple lacerations to both lids by glass and crushing against the bony orbital margins by the driven ball and a deep cut traversing the whole cornea, through which vitreous and torn iris tissue extruded, were found. There was no vision. Fragments of glass were removed, lid wounds adjusted, and the patient was sent directly to the hospital for antitetanic serum and enucleation. The eye was too badly lacerated and crushed to attempt to save it.

End-Result:

After consultation, evisceration was done on the second day, buttonholing the posterior sclera and

removing 4 mm. of the optic nerve. The remaining scleral rim with attached muscles made a better stump than implanting a glass ball in the presence of so much trauma. Deafness in one ear and general nervous instability retarded convalescence. A well-fitting and well-matched prosthesis is appreciatively worn by the patient.

CASE 7. Retained foreign body within the vitreous.

V., a plumber, aged twenty-two, while trying to remove the cap from a trap under a dirty sink with a cold chisel and hammer, was struck by "something" in his left eye, February 19, 1934. No glasses were worn. Immediate care was given by the local physician called by his employer and within two hours he was seen at the hospital. Observation showed a slight injury about the middle of the upper left lid on the margin, a penetrating wound of the cornea near the limbus at eleven o'clock, a slight tear in the pupillary margin of the iris and a hazy line through the upper third of the lens as if a foreign body had traveled from above downward toward the lower anterior vitreous body. The haziness of the lens within two hours after injury illustrates the effect and rapidity of trauma to this delicate structure. A test by hand magnet gave no response. Localization by x-ray showed a foreign body, ¾ mm. x 1¼ mm., lodged in the lower anterior vitreous chamber. The body of the trap being cast iron and the top (upon which he was chipping) being brass, the foreign body from this source would not be magnetable.

He returned home to make plans to enter the hospital for care. Antitetanic serum was given. On the third day, under local anesthesia, a giant magnet was tried. After several tries, a bright metallic foreign body was brought around the lens below and dropped on the iris below the pupillary border. The patient was placed upon a table and under the usual aseptic technique for intraocular operations, a keratome incision was made under a conjunctival flap at six o'clock and the foreign body was removed by hand magnet. This chip of magnetable steel came from the hammer or cold chisel and was probably sterile by heat when entering the eye.

He left the hospital on the twelfth day and was told to use homatropine, 2 per cent, twice daily, following a control of hypertension caused by the rapid swelling of the traumatic cataract. April 17, a needling of the anterior capsule was done to permit absorption of the soft lens mass through the aqueous. The lens was absorbed by September and a needling of the posterior capsule was made to improve vision.

Had he worn glasses or protective goggles at his work, the foreign body would never have reached his eye.

End-Result:

At the end of nine months a correcting lens gave normal vision at both distance and near, but he could not wear the strong lens necessary to accomplish this after the cataract was absorbed because of confusion to his normal right eye. The eye had begun to turn outward from disuse. The vision, however, even without any glass, was helpful to him—the peripheral vision detecting automobiles and other moving objects approaching from the left.

CASE 8. Penetrating foreign body through the cornea lodging in the lens; traumatic cataract.

T., laster in a shoe factory, aged thirty-four, slowly became aware of lacrimation of the left eye and dimness of vision. Seen first March 5, 1932, when vision was reduced to hand motion at three feet because of hazy lens, in which could be seen a small chip of metal. There was a small scar in the cornea through which the chip had entered. He was unaware of any eye injury but on careful questioning recalled that about two weeks previously he had to wipe "tears" from his eye one day while at his machine, in the operation of which small metal staples were used. He had made no report to his foreman and there developed great difficulty in the acceptance of liability by the industrial insurer. The clinical history could develop no other incident to explain the circumstances.

On October 2, the slit lamp showed a reduction in the size of the foreign body and rusty pigment diffused through the soft lens substance (siderosis). One month later a cataract operation was done, recovering the residual of the foreign body with it.

End-Result:

Three weeks later he obtained 20/30 and Jaeger No. 1 vision with strong correcting lenses but could not wear this correction without confusion to his other nearly normal eye. A glass was prescribed with instruction to wear it for protection against a similar injury in the future.

CASE 9. Penetrating wire wound through lower lid into the vitreous chamber; endophthalmitis.

M., buffing wheel maker, aged thirty-four, was struck in the left eye August 26, 1935, by a wild wire in the process of winding a buffing wheel. Immediate care was given by the factory physician. Three days later the eye was found to be red with the pupil slightly dilated by atropine, iris muddy, hypopyon in the lower aqueous chamber and vision reduced to hand motion at three feet. A wisp of vitreous extruded from an invisible (?) wound in the lower sclera. The fundus structures were not seen because of extensive exudate over the lens and haziness of the vitreous.

He was hospitalized but was not given antitetanic serum because he had had 1500 units of this given three weeks previously for a cervical wound and had showed a serum reaction. A blood count showed 12,800 white cells with 78 per cent neutrophiles. The Wassermann was negative.

Atropine frequently instilled would not keep the pupil open and suprarenin bitartrate was added twice daily, always widely dilating for a time. Sodium salicylates in 15 grain doses were given four times a day.

On September 11, Dr. Hugo Riemer was asked to see him for the insurance company and considered enucleation for safety to the good right eye, but first advised foreign protein in the form of diphtheria antitoxin daily for one week. Consulting with Dr. Sanford Hooker, immunologist of the Evans Memorial Hospital, the following procedure was carried out: After a negative intracutaneous test for sensitivity, diphtheria antitoxin was given. First, 1.5 cc., then 3 cc., and then 5 cc. daily until eight doses were given. After the eighth dose, a mild urticarial reaction was manifest and controlled by .5 cc. of adrenalin. The following dose was reduced to 3 cc.

He left the hospital on the twenty-sixth day after injury with an almost white eye and a few iris adhesions. He continued treatment as an ambulant and was instructed to return to work on November 21, having vision of 8/200 because of organized exudate over the anterior lens capsule from the excessive plastic iritis and some anterior vitreous opacities.

End-Result:

February 12, 1936, vision was 20/200, the eye white. Glasses were given for a slight refractive error in the good right eye and for protection. The eye will be kept under observation for possible

flare-ups before final insurance settlements are made.

CASE 10. Penetrating corneal wound by wire; (purulent) panophthalmitis.

Mrs. F., aged sixty-four, while shaking a rug through an open window September 21, 1929, was struck in the right eye by "something". Five hours later her physician removed a half-inch of rusty screen wire projecting from the cornea. When seen two days later, the lids were red and swollen, the eyeball was red with marked conjunctival chemosis and the anterior chamber was filled with pus. There was much pain. The patient refused operation, either enucleation or incision for drainage, consequently she was made as comfortable as possible with hot compresses, antiseptics, and so forth.

End-Result:

Phthisis bulbi (shrunken globe), no vision. Left eye is normal with glasses.

SUMMARY AND CONCLUSIONS

When one is confronted with an eye injury, the following points should be borne in mind:

1. Immediate or early care, regardless of the extent of injury, is always advisable.
2. Careful and early surgical repair of torn lids will insure best function and fewer scars.
3. X-ray to complete a diagnosis and a giant magnet to remove deep foreign bodies are essential equipment in many cases.
4. Infection is usually apparent early and is introduced, with few exceptions, at the instant of penetrating wounds.
5. Consultation with an eye surgeon or among eye surgeons is sound practice.
6. Careful watching and efficient therapeutics are imperative if the injured eye is to be saved and sympathetic involvement of the fellow eye prevented or controlled.

REFERENCES

Verhoeff, F. H.: Concerning magnetic intraocular foreign bodies and their removal. Am. J. Ophth. 15:685 (Aug.) 1932.
Woods, A. C.: Sympathetic ophthalmia. Am. J. Ophth. 19:9 (Jan.), 100 (Feb.) 1936.
Wurdemann: Injuries of the Eye. Cleveland Press.

RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS FOR JUNE, 1936

Disease	June, 1936	June, 1935	5 Yr. Average*
Anterior Poliomyelitis	6†	5	6
Chickenpox	893	1051	1052
Diphtheria	20	34	99
Dog Bite	1334	1327	986
Epidemic Cerebrospinal Meningitis	15	2	5
German Measles	684	5898	1298
Gonorrhea	406	526	591
Lobar Pneumonia	341	272	222
Measles	4066	1486	2704
Mumps	1232	406	597
Scarlet Fever	725	742	957
Syphilis	423	418	411
Tuberculosis, Pulmonary	259	312	342
Tuberculosis, Other Forms	52	26	41
Typhoid Fever	12	6	10
Undulant Fever	1	3	2
Whooping Cough	390	361	646

*Based on figures for preceding five years.
†Nonparalytic cases.

RARE DISEASES

Actinomycosis was reported from Marblehead, 1.

Anterior poliomyelitis (nonparalytic) was reported from Cambridge, 1; North Andover, 1; Southborough, 3; Worcester, 1; total 6.

Anthrax was reported from Peabody, 1.

Diphtheria was reported from Boston, 8; Cambridge, 1; Canton, 1; Chelsea, 1; Fall River, 1; Lowell, 1; Medford, 1; Newbury, 1; Salem, 1; Somerset, 1; Stoneham, 1; Taunton, 1; Worcester, 1; total, 20.

Dysentery, bacillary was reported from Attleboro, 8.

Encephalitis lethargica was reported from Lowell, 1; Milton, 1; total, 2.

Epidemic cerebrospinal meningitis was reported from Arlington, 1; Boston, 6; Brockton, 1; Deerfield, 1; Everett, 1; Fall River, 1; Fitchburg, 1; Lynn, 1; Worcester, 2; total, 15.

Malaria was reported from Winchester, 1.

Septic sore throat was reported from Barnstable 1; Belmont, 1; Boston, 7; Braintree, 1; Gardner, 1; Haverhill, 1; Lynn, 1; Petersham, 3; total, 16.

Tetanus was reported from Brookline, 1; Lee, 1; Saugus, 1; Worcester, 2; total, 5.

Trachoma was reported from Boston, 3; Brookline, 1; Chelsea, 1; total, 5.

Trichinosis was reported from Boston, 1.

Undulant fever was reported from Pittsfield, 1.

Diphtheria had its lowest reported June incidence with less cases reported for the month than might have been expected in one day in 1923.

The reported incidence of anterior poliomyelitis was not remarkable.

Epidemic cerebrospinal meningitis was reported to a higher level than any time since 1930.

A focus of canine rabies has developed in Shrewsbury and neighboring communities with eight positive heads reported through July 23.

The reported incidence of lobar pneumonia and mumps was higher than for any other June on record.

Measles, typhoid fever, and tuberculosis other forms were reported above the five-year average.

The incidence of scarlet fever, whooping cough, German measles, and chickenpox was not remarkable.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 22331

PRESENTATION OF CASE

First Admission. A thirty-three year old Canadian businessman was first seen complaining of dyspnea and swelling of the abdomen.

The patient had always been well and active until fifteen months before he was first seen, at which time he had an attack of "grippe" associated with some coryza. About a week later he had some substernal oppression and fever and a few days later a pericardial friction rub was heard. After three days a pericardial tap was done and 20 cubic centimeters of bloody fluid was removed. Subsequently he developed dyspnea and a paroxysmal cough which were relieved at intervals during the year preceding his entry by left thoracic paracentesis. Such taps produced as much as 2,000 cubic centimeters of clear straw-colored fluid which was negative for tubercle bacilli both by direct examination and guinea pig inoculation. He remained in bed for eight months, up to five months before admission, at which time he was allowed up and did well for a few weeks. During the first four months while in bed he had some fever. Three months before entry his abdomen began to swell and he again became dyspneic. Two abdominal taps, during the ten days before he was first seen, revealed large quantities of clear yellowish fluid. A month before coming to the hospital he developed gradually increasing edema of the lower extremities, trunk, neck, and face. This was associated with a varying degree of cyanosis. Throughout his illness he had a low pulse pressure and a paradoxical pulse. Another pericardial tap ten days before entry produced bloody fluid. There was little precordial pain after the first few weeks of his illness and the cough had completely subsided. He was known to have had a large liver for about one year and there was said to be a constant albuminuria. His treatment had consisted of limited activity, fluid restriction, a grain of digitalis daily, and some salyrgan, all of which were said to be effective to a certain extent.

The patient had had diphtheria in childhood, a tonsillectomy at the age of nineteen, and a left-sided dry pleurisy three years ago.

Physical examination showed a fairly well-developed and nourished, slightly cyanotic young man who appeared to be moderately ill. The eyes showed slight exophthalmos but were otherwise negative. There was faint reddening of the throat and the cervical veins were distinctly engorged. Breathing was not labored. The apex impulse of the heart was weak but was seen and felt in the fifth interspace, 10 centimeters to the left of the midsternal line. The sounds were regular and of fair quality. The second pulmonic sound was split and there was a slight to moderate paradoxical character to the pulse. A faint midsystolic murmur was heard at the apex and left side of the xiphoid in both recumbent and erect positions. Some evidence of fluid was elicited in the left chest but the remainder of the examination of the lungs was negative. The blood pressure was 110/85 although a month previously it had been 90/80. The liver was enlarged to 9 centimeters beneath the costal margin and there was some evidence of ascites. There was edema of the lower extremities and the sacrum.

The temperature was 98°, the pulse 110. The respirations were 28.

Examination of the urine showed a specific gravity of 1.020 with a trace of albumin. The sediment contained an occasional red blood cell, white blood cell, and granular cast. The blood showed a red cell count of 5,900,000, with a hemoglobin of 100 per cent. The white cell count was 15,300, 83 per cent polymorphonuclears. The nonprotein nitrogen of the blood was 40 milligrams. An electrocardiogram showed normal rhythm with low amplitude of Q-R-S and T waves in all four leads. ST_2 and ST_3 were sagging in character. A venous pressure determination was 24 centimeters of water.

X-ray examination showed dullness of the left side of the chest except for a small area in the costophrenic angle. The diaphragm on this side appeared to be low but the heart and mediastinal contents were not displaced. In the upper portion of the opaque shadow there was an irregular mottling and on the right side there was a bulge to the right in the region of the left auricle. The right lung field was clear.

On the second hospital day a pneumothorax pocket in the left chest was tapped and 275 cubic centimeters of air was removed. The paradoxical pulse became less evident and the patient's color improved. On the same day a pericardial tap was performed and 415 cubic centimeters of bloody fluid was removed. In inserting the needle a tough parietal pericardium was encountered. No organisms were found in a direct smear of the fluid. The patient responded with considerable improvement to these measures and his pulse gradually diminished to 80, the venous pressure fell, but the blood pressure and pulse pressure did not change

remarkably. His temperature, however, fluctuated between 99° and 101°. On the eighth hospital day another x-ray showed that the area of pneumothorax had disappeared and the entire left lung field was dull throughout. The heart and mediastinal contents were not displaced and the bulge to the right was smaller than when previously noted. A week later 2,090 cubic centimeters of turbid greenish yellow fluid was removed by abdominal paracentesis. The patient was discharged on the following day, to remain under observation at a local hotel.

Final Admission, six weeks later.

Five days after his discharge about 90 cubic centimeters of clear yellow fluid was removed from his left pleural cavity. Diuretics were administered with excellent results. The patient continued to run a low grade fever and on the fifteenth day after discharge examination showed that the puffiness of his face was considerably less. The veins of the neck and arms were again engorged and the venous pressure was higher (about twice the normal). The heart sounds were somewhat distant but their quality was fundamentally unchanged. There were frequent extrasystoles. The pulse was paradoxical and the blood pressure 90/65. The entire precordial area and left lower thorax was dull to percussion as was the left back up to the angle of the scapula. There was diminution in the amount of ascites. Subsequently his temperature gradually showed an afternoon rise to 102° and occasionally to 103°. Several pericardial taps were done and decreasing amounts of bloody fluid obtained. The patient now obtained less relief from these taps and diuretics likewise produced less effect. He developed a dry, annoying cough, and occasionally had a sharp shooting pain in the region of his heart. Physical signs were relatively unchanged except that a sharp clicking sound heard well with the unaided ear was elicited over the precordium during systole. Pericardial tap now showed chocolate-colored fluid with considerable pus and fibrin. No tubercle bacilli were present on direct smear. For about one week before admission the patient began to have brief periods of confusion and became evidently weaker.

He gradually became stuporous and on the day of the second admission his temperature rose to 105° and the spleen became palpable. He became comatose and died four days later, two months after his first entry to the hospital. An x-ray examination shortly before his death showed no changes in the left chest. There was, however, a diffuse mottling throughout the entire right lung.

Differential Diagnosis

Dr. Gerald Blake: We are dealing here with a man of thirty-three whose past history makes him a possible candidate for rheumatic heart disease and a more probable candidate for tuberculosis. He is said to have had grippe at the onset of his illness and grippe has come to mean any upper respiratory or sinus or respiratory infection or even the start of any disease that causes malaise with some fever. It originally was synonymous with influenza and the significance here might be that influenza tended to light up quiescent tuberculous processes. This man had a pericardial effusion that was bloody in character and while occasionally such fluid may be found in severe nontuberculous or uremic conditions we at once think of the probability of malignant disease—including lymphoblastoma—and of tuberculosis as the most likely causes. His other symptoms and signs seem to be dependent upon the presence of adhesions or constant fluid or both in the pericardium and we have evidence that both were present most of the time. A striking thing in his case is the presence of fluid in the left chest without fluid in the right chest, as is more commonly seen in failure of the heart from whatever cause. Since we do not know whether this was an exudate or transudate it may be a result of his previous pleurisy rather than a result of the incompetence of his heart. The negative guinea pig test of the fluid is against this. Enlargement of his liver and ascites is best explained on the known pericardial condition and the presence of constant albuminuria can be due to chronic passive congestion with or without some element of nephritis. The recent development of edema of the extremities, together with edema of the trunk, neck and face, may be the result of increasing interference with the venous return to the heart or possibly to more marked kidney involvement. My comment on his treatment would be that with such signs of failure and fluid he could have had more complete rest, larger doses of digitalis and more salyrgan.

The enlargement of the cervical veins found on physical examination again points to interference with the venous return either from congestive failure or from pericardial adhesions around the veins or from the pressure of a mediastinitis due to whatever caused the pericarditis. His slight degree of exophthalmos might have been due to this—or natural. I can find no other cause for it. This degree of obstruction was not sufficient, apparently, to cause labored breathing. The apex impulse shows the heart not to be much, if any, enlarged to the left and the sounds do not suggest the presence of endocarditis. The split second pulmonic sound is not significant in the presence of pericardial pressure and we should expect a paradoxical character to the pulse. I would interpret the faint midsystolic murmurs in the mitral region as an evidence of some

relative mitral regurgitation or possibly a pericardial or pleuropericardial rub. The absence of fluid in the right chest is difficult to explain. Certainly the presence of true congestive failure as a result of the pericarditis is practically ruled out by this finding, and it signifies the absence of pressure on the azygos vein on that side as well as the absence of constriction around the right pulmonary veins on entering the pericardium. The blood pressure readings were what we would expect in the presence of pericardial effusion, and the edema of the legs due to the circulatory failure and the ascites. At entrance he had no fever.

Laboratory work shows a urine which concentrates well and with its sediment findings is consistent with a considerable degree of chronic passive congestion. The findings are against true nephritis, except that the nonprotein nitrogen is slightly elevated. One may expect a secondary polycythemia of this degree with recurring fluid in the left chest and pericardium. The white count of 15,000, 83 per cent polymorphonuclears, signifies some degree of inflammatory process, but does not help us in the diagnosis. The electrocardiogram is consistent with pericardial effusion and nothing else, and the increase in venous pressure is due to either failure or interference with the venous return to the heart.

The first x-ray shows fluid pretty well throughout the left chest which does not displace the heart or mediastinum; either because of the thinness of the layer of fluid or because the mediastinal contents and heart are fixed in position by an inflammatory or malignant process. The clear area in the costophrenic angle may be due to encapsulation of the fluid above it or the presence of a little pneumothorax held at this point by encapsulation above it. The low diaphragm is in favor of this last. The irregular mottling in the upper part of the opaque shadow sounds like either metastasis or tuberculosis. If the bulge to the right is the left auricle we must consider a mitral stenosis which we have no evidence for or assume that the bulge is due to a gland or other malignant tuberculous manifestation.

The tapping of a small pneumothorax pocket on the second day improved the patient's color and pulse, again suggesting that pressure was relieved from a fixed heart and mediastinal contents. The toughness of the parietal pericardium is consistent with either of the two types of pericardial involvement we are considering, malignant disease or tuberculosis, and is also consistent with Pick's disease in which, however, we would not expect to find bloody fluid. Increase in temperature may be due to infection of the fluid somewhere or to the increase of an inflammatory or malignant process. With the whole left chest dull and no displacement of the heart the evidence is of fixed heart and mediastinal contents or little fluid or a diffuse process in the lung itself. The abdominal fluid at this time is infected from the disease process with mixed infections or possibly from repeated taps.

At his last admission his chest fluid was still uninfected although he had increase in temperature with an afternoon rise consistent with tuberculosis. There is now less space in the pericardial cavity and less relief from pericardial taps. His cough is nonproductive and the result of pleural or mediastinal irritation. I would interpret the clicking sound as due to a pericardial or pleuropericardial adhesion. The chocolate color of the last pericardial tap should be due to a combination of blood and pus. And I believe he died from septicemia superimposed on a tuberculous or malignant pericarditis which at the last became completely adherent—tuberculosis of the lungs or metastasis to both lungs, cirrhosis of the liver with perihepatitis or perisplenitis, and chronic passive congestion of the kidneys.

I will have to wait until we see the last x-ray before making the diagnosis between these two conditions from the appearance of mottling throughout the entire right lung.

At present my first choice is tuberculosis starting in the left pleural cavity; my second, malignancy starting in the left lung.

DR. AUBREY O. HAMPTON: This is the first chest film. It was taken at a relatively close distance with the patient apparently on his back. The right side of the diaphragm and the right lung are perfectly normal. The left side shows the homogeneous dulness described and the increase of radiance at the base. Since we know he had a pneumothorax and was lying on his back I think this shadow is an air bubble in the pleural cavity. It is not very characteristic of pneumothorax but I think this may be due to the position the patient was in when the film was taken. The shadow, described as bulging toward the right above the usual position of the right auricle, certainly simulates left auricle seen to the right of the spine, its lower portion, not its upper portion. Here it is much too high and is very prominent. We find very few left auricles that are so prominent, even in advanced mitral stenosis. I doubt if we should have called this left auricle. It is a queer shadow. I do not know just what to make of it.

Here is the film taken seven days later. It looks a little more like the left auricle. It is not quite so prominent.

DR. ROBERT E. GLENDY: Four hundred cubic centimeters of bloody fluid had been removed from the pericardium before that film was taken.

DR. HAMPTON: That would explain the reduction of size in the shadow and you can then say there was probably an adhesion to the peri-

cardium at this point with saeculation. The air in the pleural cavity has not disappeared. The quantity of fluid on the left side has increased. The right lung is still clear.

In this film taken two months later we still have a little air in the left pleural space, and the right diaphragm is less distinct than normal. The right border of the heart is not more brilliant than before and we have these fine miliary-like diffuse changes in the right lung which look like early miliary tuberculosis. I do not see any reason to change the diagnosis from that.

DR. GLENDY: I just want to mention a few of the salient points in this case. One of the most striking things, at least from the diagnostic point of view, is the fact that he was so much improved clinically by the removal of fairly large amounts of fluid from the pericardium, without any remarkable change in the blood pressure or pulse pressure. The cyanosis diminished, the venous pressure went down, his appetite improved, and the cough that he had disappeared. It was very striking and yet there was practically no change in his blood pressure, indicating that there was probably a constrictive process preventing adequate cardiac filling as well as cardiac tamponade from the pericardial effusion.

In regard to diuretics, I think he probably had too much salyrgan. He had been given frequent injections from which he sometimes got quite marked reactions, mainly chilly sensations and a rise in temperature. I had never seen a case in which salyrgan produced a reaction until one case preceding this, also in a patient with constrictive pericarditis.

In regard to etiology, in the cases of constrictive pericarditis that Drs. White and Churchill have reported, those with tuberculous pericarditis have done very poorly, and two who came to operation either died on the operating table or shortly thereafter. This case did not come to operation, but was under observation with that possibility in mind. In regard to the etiology of pericarditis in general, the textbooks report that perhaps tuberculous pericarditis is next in frequency to rheumatic pericarditis, but it certainly must be questionable as to whether or not many cases of uremic pericarditis are overlooked clinically. There are three forms of tuberculous pericarditis; first, that which is part of a general miliary process; the second, the diffuse form where all the serous membranes are involved to a greater or lesser extent; and the third, a dry insidious process in which adhesions usually form. I think perhaps this case is a combination of the polyserous and the adhesive types, miliary tuberculosis developing as a terminal event. Some of the largest pericardial effusions on record are the result of tuberculous pericarditis. There is considerable doubt as to whether tuberculous pericarditis can occur as a primary focus; probably not. It is remarkable, I think, that it rarely ever arises as a complication of pulmonary tuberculosis. The left pleural space of the mediastinal lymph glands must have been the primary focus here.

We did not find digitalis very useful in this case. Tapping the pericardium or the chest improved him much more than using any drug. The exophthalmos I think was partly natural and partly due to congestion. He also had considerable edema of the conjunctivae. The precordial murmur, I believe, was pericardial in origin. The absence of fluid in the right chest is difficult to explain; sometimes it is there and sometimes it is not, and occasionally it may come and go alternately on the two sides. The blood pressure readings I have already discussed. The urinary findings were probably due to congestion more than anything else.

DR. DONALD KING: I saw this patient the day before he died. There was one helpful point in the history that was not given Dr. Blake. One of the early taps did show tubercle bacilli; so we had that to go on. At that time he was said to have shown a picture of generalized tuberculosis.

DR. GLENDY: The original effusion showed tubercle bacilli in the smear but no organisms were found in smears from any of the numerous taps done here. However, the guinea pig inoculations which were reported subsequent to his death were positive.

DR. KING: With his high fever and large spleen and this x-ray picture consistent with miliary tuberculosis, it seemed that he had a generalized tuberculosis from which he was dying.

CLINICAL DIAGNOSES

Pericarditis, tuberculous.
Miliary tuberculosis.

DR. GERALD BLAKE'S DIAGNOSES

Pericarditis, tuberculous.
Miliary tuberculosis.

ANATOMIC DIAGNOSES

Pericarditis, tuberculous.
Miliary tuberculosis, generalized, involving the lungs, spleen, kidneys and adrenals.
Perisplenitis, localized.
Perihepatitis, localized.
Emaciation, moderate.
Cirrhosis, ? cardiac.
Pleuritis, left, chronic fibrous.

PATHOLOGIC DISCUSSION

DR. TRACY B. MALLORY: I do not think we concealed from Dr. Blake any information actually obtained in this hospital. The origi-

nal tap was done in Ohio and was said to have shown tubercle bacilli.

Our experience with tuberculous pericarditis is that it is often quite difficult to prove and we almost invariably are forced to resort to guinea pig inoculation to do it. At the autopsy table all anatomical evidence of tuberculosis may be lacking, even microscopic tubercles impossible to find, and only the guinea pig can be counted upon to prove the case.

He had a very extensive pericarditis with marked thickening of the pericardial walls, in places eight millimeters thick. There was still a great deal of shaggy fibrinous, quite gelatinous exudate. The fluid in the various semi-encapsulated cavities that were present was clear and showed no evidence of hemorrhage.

DR. MALLORY: The cause of death was a very widespread miliary tuberculosis in every organ of the body. The greatest surprise at the autopsy was that both adrenals were almost completely destroyed and he must have been very near to the appearance of Addison's disease. I personally do not find the lack of pleural fluid in the right chest so remarkable as the clinicians seem to think. In a chest obliterated by adhesions as in this case you cannot have a large pleural effusion. The left pleura was only partially adherent and in the unobliterated spaces he had several independent collections of fluid.

DR. BLAKE: Can you explain the round shadow in the heart findings? It suggests distortion of the pericardium.

DR. MALLORY: I think it is that. The only other thing we found that could have produced a round shadow was a very large, old bronchial lymph node but that would have been higher.

DR. KING: Would you suspect that the right pleural cavity was obliterated?

DR. HAMPTON: No, I was surprised to hear Dr. Mallory say that the right diaphragm was perfectly smooth. Usually you can see thickened pleura.

CASE 22332

PRESENTATION OF CASE

A seventy year old white woman was admitted complaining of increasing constipation.

About two and a half months before entry the patient noted a sudden change in her bowel habits. Previously she had had regular daily movements without the use of cathartics, but at this time movements required considerable straining and occasionally she had no stool for two days. She began to take laxatives at irregular intervals and about two weeks before coming to the hospital the stools became darker in color although no blood was recognized. She recalled that about six or seven months before coming to the hospital the stools were narrow and pencil-like in appearance but subsequently they became quite copious and remained so until the onset of her current illness, when they became extremely scanty except for an occasional large normal appearing movement. There was no pain until three days prior to entry, at which time a few "gas pains" occurred. There were no bowel movements for four days and enemas brought no return. The abdomen gradually became distended and she became slightly nauseated but did not vomit.

Physical examination showed a very thin, pale, elderly woman in no acute discomfort. The lungs were clear and the heart normal. The blood pressure was 130/80. The abdomen was markedly distended and tympanitic and there was slight edema of the abdominal wall. Peristalsis was not visualized but high pitched tinkling sounds could be heard with the stethoscope. No evidence of ascites was demonstrated.

The temperature was 98°, the pulse 100. The respirations were 20.

Examination of the urine showed a slight trace of albumin and a brown precipitate with the Benedict test. Large amounts of acetone were present. The sediment was negative. The blood showed a red cell count of 3,900,000, with a hemoglobin of 70 per cent. The white cell count was 20,300, 88 per cent polymorphonuclears. A stool specimen gave a positive reaction to the guaiac test. The nonprotein nitrogen of the blood was 40 milligrams. The serum chlorides were 94 cubic centimeters and the serum protein 4.3 grams. A Hinton test was negative.

A plain film of the abdomen showed a large quantity of gas in the colon and ileum. Gas in the colon extended down to the sigmoid and the cecum and terminal ileum were markedly dilated.

On the second hospital day a cecostomy was done. The patient felt quite shaky postoperatively and had a fever up to 101° for four days. Thereafter she improved considerably.

Two weeks later a barium enema ballooned the rectum and sigmoid and met a definite obstruction in the region of the junction of the descending colon and sigmoid. Barium could not be forced beyond this point. An irregular soft tissue tumor about the size of an orange was seen in this area. There was an area of calcification 2 by 1 centimeter in the left side of the true pelvis medial to the soft tissue tumor.

A week later another laparotomy was performed.

DIFFERENTIAL DIAGNOSIS

DR. HENRY H. FAXON: This is a story of subacute obstruction of the large bowel finally becoming complete in a diabetic woman

of seventy. The chief problem in differential diagnosis is whether the primary lesion was within the lumen of the bowel or whether the obstruction was caused by extrinsic pressure. The woman's age together with the lowered red count and hemoglobin make cancer as the fundamental lesion the most likely possibility.

In favor of cancer at the rectosigmoid junction are the law of probability, the characteristic story of increasing large bowel obstruction, and the finding of a positive guaiac in the stool on the only examination quoted. However there are certain definite objections to this diagnosis which make me exclude it as my first choice.

In the first place the history tells us that there was a "sudden" change in her bowel habit only two and a half months before entry. With carcinoma of the large bowel that had gone on to complete obstruction an antecedent history dating back a longer time would be more characteristic. If we evaluate the story further it may be that a longer period of difficulty was actually present, for below the original statement comes the remark that "about six or seven months before coming to the hospital the stools were narrow and pencil-like in appearance but subsequently became quite copious and remained so until the onset of her current illness, when they became extremely scanty except for an occasional large normal appearing movement." If her obstruction were due to a lesion arising in the large bowel it seems improbable to me that she would have experienced such freedom from symptoms between the time mentioned seven months before and the later date of two and a half months prior to entry which she gives for the onset of her trouble. It is not inconsistent with an obstructing lesion of the colon to have "copious" movements, for if the pathology is high enough in the colon the rectum can accumulate a good deal of feces below the obstruction before the person is actually called upon to defecate. The absence of tenesmus in this case is clinical evidence that the lesion was not low, which is later confirmed by the site of obstruction as demonstrated by x-ray.

The second point which seems unusual for a lesion primary in the large bowel is the relative absence of blood in the stools. It is true that some blood was present because the guaiac was positive and the patient noted that the stools had become somewhat darker. But this observation had been made only two weeks before coming to the hospital. The patient had never recognized any blood in the stools and the degree of anemia was not severe.

In the third place there is a note that not only was the abdomen distended and tympanitic but also that there was "slight edema of the abdominal wall" at the time of complete obstruction. It would be most unusual in the absence of ascites or any note of edema elsewhere to have an intrinsic lesion of the large bowel giving rise to edema of the abdominal wall.

In the last place the x-ray showed an "irregular soft tissue tumor" in the region of the obstruction, with an area of calcification on its medial side, which would be an unusual report for a lesion arising within the bowel.

The laboratory data other than those which have already been commented upon give little added information save for the fact that she apparently had diabetes with associated acidosis. In the face of a normal temperature I should ascribe her white count of 20,000 to her diabetic acidosis, and I do not believe that the factor of infection was of primary importance.

The cecostomy apparently relieved her acute obstruction and her temperature postoperatively could easily be accounted for on the basis of the unavoidable soiling that the operation occasioned. Diverticulitis might be mentioned in connection with a lesion in this region. However, no diverticula were seen in the large bowel as visualized by x-ray, and bleeding which we know had occurred would be most unusual in diverticulitis.

The area of calcification seen by x-ray most probably was either in the ovary or in a fibroid. The presence of a pelvic tumor might easily have been missed on examination due to the degree of distention from the large bowel obstruction which was present at the time the examination was made.

My diagnosis in this case is diabetes and obstruction of the large bowel at the rectosigmoid junction. I think that the obstruction was caused by a malignancy extrinsic to the large bowel but in part invading and involving it. The most probable origin of this malignancy would be found in the pelvic organs, most probably a fibroid of the uterus that had undergone malignant degeneration.

Clinical Diagnosis

Carcinoma of the sigmoid.

Dr. Henry H. Faxon's Diagnosis

Obstruction of the sigmoid from extrinsic neoplasm, probably sarcoma of the uterus.

Anatomic Diagnoses

Adenoacanthoma, probably primary in the sigmoid, with invasion of the left ovary and metastasis to the lung.
Septic infarction of the lungs.
Retroperitoneal abscess.
Emaciation.
Operative wounds: Cecostomy, colo-sigmoidostomy.

Pathologic Discussion

Dr. Tracy B. Mallory: The preoperative diagnosis in the hospital was carcinoma of the sigmoid. The patient was in such an emaciated and dehydrated state that the first operation consisted only of a cecostomy done under local anesthesia; no attempt at exploration was made. Two weeks later she was operated upon again, this time through a left rectus incision. A tumor mass was found which tied together the upper end of the sigmoid, the ovary, and the uterus. A note was made that the broad ligament was invaded. It was felt that resection was out of the question so an anastomosis short-circuiting the growth was done between the descending colon and the sigmoid below the tumor. It was still felt that the tumor was presumably primary in the bowel. Following operation the patient showed some evidence of wound sepsis and eventually developed bronchopneumonia and died.

As Dr. Faxon pointed out in his first sentence, the crux of the differential diagnosis in this case was whether the bowel was obstructed by an intrinsic or an extrinsic tumor. He placed his stakes on the latter, the surgeons who had charge of the patient in the hospital on the former. At the conclusion of the autopsy I was sure that the tumor arose in the ovary but since studying the microscopic sections I am once more in doubt and feel forced to assess the balance of evidence as slightly in favor of the sigmoid origin.

At autopsy we found the left ovary and tube completely replaced by a tumor mass five centimeters in diameter. This was adherent to and had apparently invaded the wall of the sigmoid. The mucosa of the sigmoid, however, appeared to be continuous over the tumor and showed no gross ulceration. This is so unusual for a malignant tumor of the large bowel that we had no hesitation in signing the death certificate as primary ovarian carcinoma. There was no intrinsic narrowing of the gut and the pressure of the tumor mass from without seemed certainly responsible for the obstruction, as Dr. Faxon predicted.

Microscopic examination of the tumor, however, proved at once interesting and surprising. The tumor turned out to be a so-called adenoacanthoma, that is a carcinoma differentiating in some areas into glandular epithelium, in other areas into squamous cells with epithelial pearl formation. These tumors are a well recognized entity in the endometrium and have also been noted with some frequency in the gallbladder. I have seen two of them in the lower end of the esophagus and one which was certainly primary in the large bowel. I know of no report of such a tumor arising in the ovary and am therefore inclined to swing my verdict over to a primary adenoacanthoma of the sigmoid.

The other findings of significance were a retroperitoneal abscess in the region of the intestinal anastomosis and a number of small septic infarcts in the lung. One nodule of metastatic tumor, one centimeter in diameter, was found in the left upper lobe of the lung.

The New England
Journal of Medicine
SUCCESSOR TO
THE BOSTON MEDICAL AND SURGICAL JOURNAL
Established in 1828

Published by THE MASSACHUSETTS MEDICAL SOCIETY under the jurisdiction of the

COMMITTEE ON PUBLICATIONS
R. I. LEE, M.D., *Chairman* R. M. SMITH, M.D.
R. B. OSGOOD, M.D. F. H. LAHEY, M.D.
J. P. O'HARE, M.D.

EDITORIAL STAFF
JOHN P. SUTHERLAND, M.D. STEPHEN RUSHMORE, M.D.
GEORGE R. MINOT, M.D. HANS ZINSSER, M.D.
FRANK H. LAHEY, M.D. BENJAMIN WHITE, PH.D.
SHIELDS WARREN, M.D. HENRY R. VIETS, M.D.
GEORGE L. TOBEY, JR., M.D. ROBERT M. GREEN, M.D.
C. GUY LANE, M.D. CHARLES C. LUND, M.D.
WILLIAM A. ROGERS, M.D. JOHN F. FULTON, M.D.
DWIGHT O'HARA, M.D. A. WARREN STEARNS, M.D.

ASSOCIATE EDITORS
ROBERT N. NYE, M.D. GEORGE G. SMITH, M.D.
JOSEPH GARLAND, M.D. WILLIAM B. BREED, M.D.

WALTER P. BOWERS, M.D., *Managing Editor*
CLARA D. DAVIES, *Assistant Editor*

THE NEW HAMPSHIRE MEDICAL SOCIETY
PUBLICATION COMMITTEE
CARLETON R. METCALF, M.D. HENRY H. AMSDEN, M.D.
WARREN H. BUTTERFIELD, M.D.

THE VERMONT STATE MEDICAL SOCIETY
PUBLICATION COMMITTEE
WILLIAM G. RICKER, M.D. C. F. DALTON, M.D.
L. H. ROSS, M.D.

SUBSCRIPTION TERMS: *$6.00 per year in advance, postage paid for the United States, Canada $7.04 per year, $8.52 per year for all foreign countries belonging to the Postal Union.*

Material for early publication should be received not later than noon on Saturday. Orders for reprints must be sent to the Journal office, 8 Fenway.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to The New England Journal of Medicine, 8 Fenway, Boston, Mass.

CHARLES HARRISON FRAZIER
(1870-1936)

WITH the death of Dr. Charles H. Frazier of Philadelphia another landmark in the development of the special field of surgery of the nervous system is passed. With Kocher, Macewen and Horsley as a background, the stage was set in the late nineties for great advances in intracranial surgery. Harvey Cushing and Frazier were the pioneers in this country. Cushing, straight from Kocher and Sherrington, began to cut deeper into cerebral tissue and extirpate new growths more fully than any surgeon before him, always preserving the gentle touch gained from Halsted. Frazier, on his return from Berlin, first turned to general surgery. He was soon stimulated, however, by the possibilities of surgical therapy in the many patients gathered in Philadelphia, then the center of neurology in America, under the care of the younger and older Mitchells, Mills, Spiller and others. His work on chordotomy for the relief of pain, for which he is best known, came as the result of co-operation with Spiller. Spiller worked out the location of the spinal pain tracts, suggested the operation and Frazier supplied the surgical technique.

As time went on Frazier largely gave up general surgery and devoted his time to the removal and study of brain tumors, the surgery of trigeminal neuralgia and other forms of cranial nerve disorders, brain abscess and pituitary tumors. At the same time he continued his interest in the surgery of the spinal cord and wrote the most adequate text published in the country on the subject. For many years he almost annually reported his neurosurgical results at the meetings of the American Neurological Association. Clear-cut in both writing and in speech, he left a lasting impression on readers and hearers of sound productive scholarship. His clinic grew rapidly and many students were trained by him. Something of the driving type, a number of operations a day were not an unusual occurrence. His training in general surgery never left him, even when entering the cranial cavity. His results, however, have been exceeded by few. Frazier's best work was his earliest, on spinal cord surgery. Unfortunately he never found time to bring out successive editions of his text.

EXTREME HEAT vs. HUMAN BEINGS

THE figures given in the *Weekly Health Index*, published by the Bureau of the Census, Department of Commerce, Washington, for the week ending July 18, 1936, might well be used by the New England Council as propaganda in recommending New England as a summer resort.

During the week reported, there were 12,183 deaths in eighty-six large cities compared with 7,439 in 1935, an increase of 4,744 or 64 per cent. If one considers the figures for the individual cities it is found that the average increase in mortality for mid-western cities in the extreme heat zone was over 200 per cent. Peoria had an increase of 680 per cent and St. Paul one of 400 per cent! During the same period there was an increase in mortality in fourteen New England cities of only 2 per cent, a figure well within the normal year-to-year fluctuation. It is interesting to note that in eight southern cities, where the residents are accustomed to sustained heat, the average increase in mortality was only 25 per cent.

While it is realized that many of these deaths were accidental in the course of activities, such as boating and swimming, which are increased during extremely hot weather, there is no doubt but that many human machines, normally attuned to a temperate climate, are unable to perform their functions and tasks

under conditions of prolonged and extreme heat.

The vulnerability of human beings to high temperatures is also proved by an increase in infant mortality of 34 per cent during the same week, which was accounted for, in a large part, by deaths in the heat zone. This increase is probably much more significant than the increase in general mortality, for children under one year of age are, ordinarily, much less liable to die following accidents or from chronic diseases, the courses of which are fatally terminated by extreme degrees of temperature.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

CHADWICK, HENRY D. M.D. Harvard University Medical School 1895. Formerly, Superintendent of the Westfield State Sanatorium, and Tuberculosis Controller in Detroit, Michigan. Now, Massachusetts Commissioner of Public Health. Address: State House, Boston, Mass. Associated with him is

LOMBARD, HERBERT L. A.B., M.P.H., M.D. Bowdoin Medical School 1915. Director, Division of Adult Hygiene, Massachusetts Department of Public Health. Assistant Professor of Hygiene and Public Health, Tufts College Dental School. Address: 100 Nashua Street, Boston, Mass. Their subject is The Massachusetts Cancer Program. Page 265.

KUHNS, JOHN G. A.B., M.D. Johns Hopkins University School of Medicine 1924. Chief-of-Staff, Robert Breck Brigham Hospital. Consulting Orthopedic Surgeon, Sturdy Memorial Hospital, Attleboro. Assistant Orthopedic Surgeon, Children's Hospital, Boston. Assistant in Orthopedic Surgery, Harvard University Medical School. Address: 372 Marlborough Street, Boston, Mass. Associated with him is

JOPLIN, ROBERT J. A.B., M.D. Harvard University Medical School 1929. Assistant Orthopedic Surgeon, Children's Hospital and Robert Breck Brigham Hospital. Assistant in Orthopedic Surgery, Harvard University Medical School. Address: 300 Longwood Avenue, Boston, Mass. Their subject is Convalescent Care in Chronic Arthritis. Page 268.

MORRIS, ROBERT H. M.D. University of Toronto Medical School 1922. F.A.C.S. Assistant Orthopedic Surgeon, Children's Hospital, Boston. Instructor in Orthopedics, Harvard University Medical School. Consulting Orthopedic Surgeon, New England Baptist Hospital. Orthopedic Surgeon, Long Island Hospital. Address: 253 Newbury Street, Boston, Mass. Associated with him is

DOWNING, F. HAROLD. A.B., M.D. Leland Stanford University Medical School 1929. Assistant in Orthopedic Surgery, Massachusetts General Hospital. Assistant Visiting Orthopedic Surgeon, Long Island Hospital. Assistant in Orthopedic Surgery, Cambridge Hospital. Address: 253 Newbury Street, Boston. Their subject is Report of a Case of Vertical Fracture Through the Lower Tibial Epiphysis During the Period of Bone Growth and an Operation for the Correction of the Resultant Deformity. Page 272.

MAY, G. ELLIOTT. A.B., M.D. Harvard University Medical School 1925. Assistant Visiting Surgeon for Gynecology and Obstetrics, Boston City Hospital. Senior Obstetrician, Newton Hospital, Newton. Assistant Obstetrician, Massachusetts General Hospital, Boston. Consultant in Obstetrics, Framingham-Union Hospital, Framingham, and Anna Jaques Hospital, Newburyport. Assistant in Obstetrics and Gynecology, Harvard University Medical School. His subject is Dehydration Therapy in the Toxemias of Pregnancy. Page 277. Address: 201 Bay State Road, Boston, Mass.

HARTWELL, CONSTANCE G. S.B., M.D. Boston University School of Medicine 1935. Now an Interne at the Massachusetts Memorial Hospitals, Boston. Address: West Newton, Mass. Associated with her is

ROWLAND, WILLIAM D. M.D. University of Michigan Homeopathic Medical School 1911. F.A.C.S. Professor of Ophthalmology, Boston University School of Medicine. Chief of Eye Service, Massachusetts Memorial Hospitals. Address: 84 Commonwealth Avenue, Boston, Mass. Their subject is The Problem of Eye Injuries. Page 290.

MISCELLANY

POLIOMYELITIS IN THE SOUTH

No indications are seen by U. S. Public Health Service officials that the infantile paralysis (poliomyelitis) epidemic in Alabama and Tennessee will reach national proportions. Reports from other parts of the country show no unusual amount of the disease. Federal health authorities are also encouraged by the fact that the Alabama-Tennessee outbreak is not so severe as the North Carolina epidemic of about the same time last year. Nor does it show any great tendency to spread.

Federal "H-men" led by Dr. Charles Armstrong have gone into the affected areas in order to aid in the application of the new nose spray which it is hoped will prevent the disease. Developed by Dr. Armstrong and Dr. W. T. Harrison as the result of experimental work on monkeys, the alum-picric acid nasal spray is receiving its first large-scale application in this epidemic.

In the hope of saving some of those who might otherwise fall victims, the nasal spray is being used without any attempt at making a controlled experiment. Physicians and health officers are administering the spray, which is quite harmless, to those who desire it and who can be treated with the facilities available. Undoubtedly a study will be made later to determine whether any cases of poliomyelitis occur among those who are treated with the spray, but there is no systematic exclusion of some from the treatment in order to have a "normal" group in which the disease might have an unhampered chance to spread, as would be the case if the doctors were conducting a laboratory experiment.

U. S. Public Health Service officers are careful to state that the alum-picric acid spray method is "based entirely upon animal experimentation" and "is not at present to be regarded as of proved value in the prevention of poliomyelitis in man." Nevertheless, they are hopeful that it will prove effective in preventing the disease and that this epidemic may give some evidence to that effect.

In last year's North Carolina epidemic there was experimental use of vaccines designed to provide artificial immunity to the disease. In the time subsequent to that use medical opinion has developed which has indicated that vaccines should not be used.—*Science Service.*

STATEMENT REGARDING NASAL SPRAY AS PREVENTIVE OF POLIOMYELITIS

The recent experimental work by Drs. Armstrong and Harrison in preventing poliomyelitis in monkeys by the use of a nasal spray has excited so much interest and speculation that the Public Health Service deems it desirable to issue the following statement:

The evidence regarding this method is as yet based entirely upon animal experimentation and the proposed spray is not at present to be regarded as of proved value in the prevention of poliomyelitis in man. It may be advisable to await the results of further trials before giving the method general application. If, however, it is desired to use the solution, it should be sprayed into the nostrils three or four times on alternate days, and thereafter weekly during the presence of poliomyelitis. The spray tip should be pointed upward and backward at an angle of about 45°, and the spraying should be thorough enough to reach the pharynx as well, when a bitter taste will be noted. The early applications at least should be administered by a physician. The experimental work on animals is still being pursued. The tentative procedure is, therefore, subject to such changes as may be dictated by future findings.

The most effective solution so far developed during experimentation on monkeys is prepared as follows:

Solution A: Dissolve 1 gram of picric acid in 100 cc. of physiological salt solution (0.85 per cent). (Warming facilitates solution of the picric acid.)

Solution B: Dissolve 1 gram of sodium aluminum sulphate (sodium alum) in 100 cc. of physiological salt solution (0.85 per cent). Any turbidity in this solution should be removed by filtering one or more times through the same filter paper.

Mix solutions A and B in equal amounts. The resulting mixture, which contains 0.5 per cent picric acid and 0.5 per cent alum, is sufficiently antiseptic to prevent the growth of organisms and is ready for use as a spray. Home-made concoctions are not favored.—*Public Health Reports* 51:978 (July 17) 1936.

VIOLATORS OF FOOD AND DRUG LAW FINED

Shippers of dairy products, fish, nut meats, tomato products, fresh vegetables, macaroni and medicines, were brought before the Federal courts during June on a variety of charges, the current report of the Food and Drug Administration shows. Fines totaling $6,175 were assessed, with costs added in some instances.

Two other cases involving substandard pharmaceuticals were terminated during June, resulting in a fine of $200 to the G. W. Carnrick Co., Newark, N. J., and one of $50 to the Cheplin Biological Laboratories, Inc., Syracuse, N. Y. In Cleveland, Ohio, a federal district judge overruled a demurrer to the information charging violation of the Food and Drugs Act in the shipment of a product labeled "Epsom Salt Compound Tablets." The manufacturer — Strong, Cobb & Co., — contended that the word "compound" in the name of the drug was a sufficient notification of the inclusion of powerful laxatives that the Administration contends the purchaser would not expect. The predominantly active ingredients in these tablets were shown by analysis to be phenolphthalein (a coal-tar laxative) and an extract of the laxative drug aloe. Although Epsom salt was in fact present, the quantity of it fell far short of even a mild dose, the product actually depending for its effect upon the drug ingredients that were neither announced on the label nor indicated in the name of the preparation.

The report of the termination of one patent medicine case concludes the June statement of prosecutions involving drugs. Henry Lutzenkirchen, trading as the Ora-Noid Co., Chicago, Ill., dropped his intended contest of the government's case against him, based on a shipment of his product "Ora-Noid," and was fined $50 and costs. "Ora-Noid," half table salt and the remainder consisting of chalk, baking soda, magnesium and potassium sulphates and phosphates, flavored with an essential oil, was offered as a "complete oral prophylactic". These fraudulent claims have now been removed from the labeling of the product.—*U. S. Department of Agriculture.*

TUBERCULOSIS ABSTRACTS

The year Koch announced the discovery of the tubercle bacillus, 1882, Forlanini published his first paper on pneumothorax. Yet only during the last decade has collapse therapy become accepted prac-

tice in this country. What are the more important considerations that have contributed to this change in the treatment and management of pulmonary tuberculosis? J. W. Cutler, physician-in-charge of the pneumothorax clinic, Henry Phipps Institute, discusses this subject in an article in the *Journal of the American Medical Association* of April 18, 1936.

NATIONAL MEDICAL COUNCIL ON BIRTH CONTROL

The National Medical Council on Birth Control was organized in June, 1936, for the following purposes:

1. To control and supervise all medical policies of the American Birth Control League.
2. To initiate, encourage, and execute appropriate scientific research in the medical aspects of birth control.

EXECUTIVE COMMITTEE

Frederick C. Holden, M.D., Chairman
Eric M. Matsner, M.D., Executive Secretary
Eliot Bishop, M.D., Brooklyn
A. N. Creadick, M.D., New Haven
Foster Kennedy, M.D., New York
Edgar Mayer, M.D., New York
Richard N. Pierson, M.D., New York
Owen Toland, M.D., Philadelphia
Wilbur Ward, M.D., New York
Prentiss Willson, M.D., Washington.

THE COUNCIL

Fred L. Adair, M.D., Chicago
Brooke M. Anspach, M.D., Philadelphia
John M. Bergland, M.D., Baltimore
P. Brooke Bland, M.D., Philadelphia
Alexander M. Campbell, M.D., Grand Rapids
Elizabeth Campbell, M.D., Cincinnati
F. Bayard Carter, M.D., Durham
William H. Cary, M.D., New York
Ross Chapman, M.D., Towson
Arthur H. Curtis, M.D., Chicago
William C. Danforth, M.D., Evanston
Nathan Davis, III, M.D., Chicago
Robert L. DeNormandie, M.D., Boston
Robert L. Dickinson, M.D., New York
Nicholson J. Eastman, M.D., Baltimore
Franklin G. Ebaugh, M.D., Denver
John Erdmann, M.D., New York
John Favill, M.D., Chicago
Palmer Findley, M.D., Omaha
Morris Flexner, M.D., Louisville
Clarence Gamble, M.D., Philadelphia
Francis C. Goldsborough, M.D., Buffalo
John W. Harris, M.D., Madison
N. Sproat Heaney, M.D., Chicago
J. Shelton Horsley, M.D., Richmond
Floyd E. Keene, M.D., Philadelphia
Sophia J. Kleegman, M.D., New York
Rae T. La Vake, M.D., Minneapolis
Jennings C. Litzenberg, M.D., Minneapolis
William Palmer Lucas, M.D., San Francisco
Frank Lynch, M.D., San Francisco
Harvey B. Matthews, M.D., Brooklyn
James R. McCord, M.D., Atlanta
Lyle G. McNeile, M.D., Los Angeles
Adolph Meyer, M.D., Baltimore
Hilliard E. Miller, M.D., New Orleans
Norman F. Miller, M.D., Ann Arbor
Bessie L. Moses, M.D., Baltimore
Stuart Mudd, M.D., Haverford
F. O. Plunkett, M.D., Lynchburg
William Allen Pusey, M.D., Chicago
Albert Sparr Rider, M.D., Flandreau
Milton J. Rosenau, M.D., Chapel Hill
Edward A. Schumann, M.D., Philadelphia
William Sidney Smith, M.D., Brooklyn
Raymond Squier, M.D., New York
Kyle B. Steele, M.D., New York
Irving F. Stein, M.D., Chicago
Eric Stone, M.D., Providence
William E. Studdiford, M.D., New York
Fred J. Taussig, M.D., St. Louis
Howard C. Taylor, Jr., M.D., New York
Kenneth Taylor, M.D., New York
Benjamin Tilton, M.D., New York
Norris W. Vaux, M.D., Philadelphia
Fred C. Warnshuis, M.D., San Francisco
Ira S. Wile, M.D., New York
Philip F. Williams, M.D., Philadelphia
Tiffany J. Williams, M.D., University
Milton C. Winternitz, M.D., New Haven
Hans Zinsser, M.D., Boston.

ABSTRACTS OF TWO PAPERS READ AT THE ROCHESTER MEETING OF THE AMERICAN ASSOCIATION AND ASSOCIATED SOCIETIES

Experiments on the effect of the new neutron rays of science to produce biological changes in living organisms show that they are much more efficient than x-rays, according to Dr. Raymond E. Zirkle, Johnson Foundation for Medical Physics at the University of Pennsylvania. Ultimate hope of the investigators, Dr. Zirkle revealed, is that the neutron rays will be more destructive to tumorous tissue than to normal body tissue and that medical science will thereby have a more potent radiation with which to attack cancer. He stated that "the results to date do not justify the prediction of such an extremely fortunate outcome, but are nevertheless distinctly encouraging." The neutron—one of the fundamental building blocks out of which atoms are built—was not discovered until 1932. Even yet the scources for creating neutron beams for biological study are so weak that only beams of low intensity can be used. But the encouraging thing is that the effectiveness of neutrons in ionizing tissue (which is the way all rays act on biological material) is always greater than x-rays for equal incident intensities. The relative effectiveness has been found to be from three to ten times in favor of the neutrons. Moreover, and still more encouraging, the neutron-x-ray ratio of effectiveness is not the same

for all living tissue. "This is of tremendous importance," according to Dr. Zirkle.

New advances in studying the tuberculosis germ were reported by Professor R. J. Anderson, of Yale University. By analyzing these germs, Professor Anderson is learning what chemicals they use to produce disease in man and other animals. In wax obtained from the germ he has found complex sugars, fatty acids and a new alcohol which are peculiar to the human tuberculosis germ and are not found in waxes from any other bacteria. The part these different tuberculosis germ chemicals play in producing the disease has not been completely established, but Dr. Florence Sabin, of the Rockefeller Institute for Medical Research, has found that the new acid and the optically active alcohols of the wax from the tuberculosis germs are remarkable stimulants to cells, especially the type of cells that form fibrous tissue and tendons in the body, the white blood cells known as monocytes and foreign body giant cells.—*Science Service.*

NEW YORK CHILDREN'S SOCIETY LAUNCHES MOTHERS' MILK PRESERVING PROCESS LICENSED TO IT FREE BY BORDEN COMPANY*

Doctors Declare Death Rate of Premature Babies Can Be Cut; To Issue Other Licenses

Pediatricians and doctors in New York City—including Health Commissioner John L. Rice; Dr. Shirley W. Wynne, former Health Commissioner; and Dr. Carl H. Laws, Chief of Pediatric Service, Long Island College Hospital — saw new hope for saving the lives of an increasing number of prematurely born babies when, on July 24, the Mothers' Milk Bureau of the Children's Welfare Federation announced the adoption of a new process of quickly freezing mothers' milk.

The process was invented by Washington Platt, a scientist in the Syracuse, N. Y., research laboratory of The Borden Company, and was clinically tested by Dr. Paul W. Emerson of Boston. It makes it possible for the first time to keep mothers' milk for several months or more, transport it, and feed it to prematurely born or ill babies without any subsequent action other than thawing and warming.

A license for the use of the new process, without charge, was turned over to the Federation by The Borden Company during ceremonies on July 24. Dr. James A. Tobey, Director of Health Service of the company, made the presentation.

"We are happy to be able to contribute in this way to the welfare not only of the babies of New York City, but of the nation," Dr. Tobey said. "With an infant death rate in New York in 1935 of 47.6 per 1,000 live births and with 30 per cent of these deaths due to prematurity, every scientific step should be taken to reduce this toll of young lives. It was with this thought in mind that our company determined to license this process without charge to the medical profession so that an increasing number of infant lives might be saved."

Dr. Wynne, President of the Federation, in accepting the license, said: "We believe this process will enable us to preserve the excess milk we receive in periods when the supply exceeds the demand, thereby building up a reserve supply to cope with all emergencies. When we have had excess quantities in the past we have tried to can the milk but that is not satisfactory. This process should enable us to meet any demand that is made upon us."

Dr. Rice congratulated the Federation on its work in providing a constant supply of mothers' milk for premature and ill babies. "I am happy that this important step is being taken to reduce the mortality rate of babies in their first year of life," he said.

Mr. Platt explained the process. "It provides for the freezing of mothers' milk under such conditions that, upon thawing, the natural emulsion will remain substantially unbroken and other characteristics will remain sufficiently unchanged so that the thawed product will be suitable for infant feeding," he declared.

"The apparatus consists of moulds, into which the milk is put, and a cover. Dry ice is used to freeze the milk into wafers quickly. These are then put in sterile bottles and stored at a low temperature. The milk will keep indefinitely. It is thawed by immersing the receptacle in cold water or by allowing it to stand in an ordinary ice box until thawed. It is warmed to body temperature before feeding."

A study made by Dr. Walter H. Eddy of Teachers College, Columbia University, showed that frozen human milk as compared with unfrozen milk has lost none of its content of vitamins. Moreover, frozen human milk does not differ from fresh milk in total solids, fats, carbohydrates or proteins.—*Bureau of Industrial Service, Inc.*

Editorial Note: This process has been in use in Boston by The Directory for Mothers' Milk, Incorporated, for three years.

NATIONAL UNIVERSITY OF IRELAND CONFERS HONORARY DEGREE ON DR. FLEXNER

The National University of Ireland conferred the honorary degree of doctor of science on Dr. Simon Flexner, member emeritus of The Rockefeller Institute for Medical Research, New York, at University College, Dublin, on July 20.—*Science*, August 7, 1936.

*See the paper of Dr. Paul W. Emerson in *The New England Journal of Medicine* 209:893 (Nov. 2) 1933 which was read before the Pediatric Section of the Massachusetts Medical Society, June 7, 1933. It is not likely that the New York Group are unmindful of this priority of procedure in the use of frozen mothers' milk, since their representatives visited the Boston organization and learned about its use here before this method was adopted in New York.

COMPARISON OF DISEASE INCIDENCE IN CONNECTICUT WITH 1935 AND SEVEN YEAR AVERAGE

MONTH ENDING JULY 18, 1936

Diseases	1936 Week ending June 27	1936 Week ending July 4	1936 Week ending July 11	1936 Week ending July 18	Average cases reported for week corresponding to July 18 for past seven years	1935 Week ending June 29	1935 Week ending July 6	1935 Week ending July 13	1935 Week ending July 20
Chickenpox	70	40	34	54	22	64	52	79	38
Conjunctivitis, Infectious	—	13	13	—	—	11	—	1	—
Diphtheria	—	2	1	—	9	19	11	9	7
German Measles	92	104	48	22	3	66	33	38	21
Influenza	2	1	—	—	2	—	—	1	1
Malaria	—	1	—	—	—	—	—	—	1
Measles	49	78	53	41	68	301	223	167	70
Meningococcus Meningitis	—	—	1	—	1	—	1	—	—
Mumps	58	32	62	25	16	29	15	23	14
Paratyphoid Fever	1	1	1	—	—	4	—	1	—
Pneumonia (Broncho)	4	9	13	19	9	5	11	9	8
Pneumonia (Lobar)	16	12	17	23	8	15	12	12	3
Poliomyelitis	—	—	—	1	2	1	—	2	3
Scarlet Fever	22	14	10	7	14	50	23	33	6
Streptococcus Sore Throat	5	1	2	4	1	—	2	4	3
Tetanus	—	1	—	1	—	—	—	—	—
Trachoma	—	1	—	—	—	—	—	—	—
Tuberculosis (Pul.)	31	25	21	35	28	19	15	17	17
Tuberculosis (O. F.)	1	5	1	3	4	2	4	1	5
Typhoid Fever	2	—	4	1	2	1	—	—	2
Undulant Fever	2	4	7	4	—	1	2	2	—
Whooping Cough	42	75	40	48	53	36	22	46	38
Gonorrhea	28	41	31	25	36	47	22	32	26
Syphilis	43	80	47	37	45	94	50	45	28

Remarks: No cases of Asiatic cholera, glanders, plague or yellow fever during the past seven years.

CORRESPONDENCE

UNJUST ACTION OF INSURANCE COMPANIES

July 31, 1936.

Editor, *New England Journal of Medicine*,

I am keenly interested in Dr. Charles Malone's letter in the *Journal* of July 16, 1936, page 134. I wish to extend my congratulations to him for bringing to the attention of the medical profession the unfair way industrial accidents and diseases are handled in that the injured or diseased employee does not have a chance to choose the doctor he wishes to have treat him.

Another great unfairness that many doctors have been made to suffer, is the manner in which the insurance companies accuse us of bill padding, over-attention, and exorbitant charges, and when it comes to paying, they demand a big reduction on every bill that amounts to much and some reduction on many of the small ones.

The adjusters "so-called," but they might, perhaps, better be called intimidators, all use the stock sentence that they have no trouble adjusting bills with any other doctor, and that they all co-operate very nicely. I have found out that such is not the case. I have talked the matter over with many colleagues, and they too know as I do that we are being handled by insurance companies in a manner that is very unfair to say the least.

The time has come when men of the medical profession should get together and collectively demand fair and just treatment in the above matters.

Because we, as a profession, have always worked for the poor, free of charge, does not make it necessary for the insurance companies to expect us to extend our charity to them. The premiums which they charge to carry insurance should be sufficient to keep injured workmen from the charity class. Employed people that, because of accident or industrial disease, need surgical or medical treatment, ought to be informed of our state law pertaining to employees, and allowed, without being intimi-

dated, to select the doctor they wish to have treat them.

Yours very truly,

GEORGE J. BASSOW, M.D.

193 Main Street,
Athol, Mass.

ARTICLES ACCEPTED BY THE AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

535 North Dearborn Street, Chicago, Illinois,
August 4, 1936.

Managing Editor,
The New England Journal of Medicine,

In addition to the articles enumerated in our letter of June 30 the following have been accepted:

Lederle Laboratories
- Ash Pollen Antigen—Lederle
- Beech Pollen Antigen—Lederle
- Hickory Pollen Antigen—Lederle
- Poplar Pollen Antigen—Lederle
- Sycamore Pollen Antigen—Lederle

Sharp & Dohme, Inc.
- Tetanus Gas-Gangrene Antitoxin Mixed — Mulford

E. R. Squibb & Sons
- Gas Gangrene Antitoxin—Squibb
- Tetanus-Gas Gangrene Antitoxin—Squibb

Yours sincerely,

PAUL NICHOLAS LEECH, *Secretary*.

RECENT DEATH

McDONALD—WILLIAM MCDONALD, M.D., formerly of Providence, Rhode Island, but since 1924 a resident of Marion, Massachusetts, died at his home, August 1, 1936, after a long illness.

Dr. McDonald was born in Albany, New York, in 1873 and graduated from Columbia University College of Physicians and Surgeons in 1899. He was a Fellow of the Massachusetts Medical Society and the American Medical Association and was a specialist in Neurology and Psychiatry. When President Roosevelt was suffering with anterior poliomyelitis, Dr. McDonald was his physician.

Dr. McDonald's wife, Elizabeth Hurkamp McDonald, died in January, 1934. Three brothers survive him, Donald of New York and Hyannisport, Albert of Garden City, L. I., and Frederick of Albany, as do two sisters, Mrs. Harry Taylor and Miss Frances McDonald.

OBITUARY

SIR HENRY WELLCOME

In his eighty-third year Sir Henry Wellcome died in London on July 25 following an operation. Born in 1853 in a log cabin near Almond, Wisconsin, about 125 miles from Milwaukee, he was the son of an itinerant missionary, the Rev. S. C. Wellcome who with his wife, Mary Curtis Wellcome, traveled in a covered wagon in Wisconsin and Minnesota and preached among the Indian settlements.

At an early age he began his career as a pharmacist in Rochester, Minnesota, where he worked from 1868 to 1871. It was there that he came under the notice of Dr. William Wardell Mayo, father of Dr. William J. Mayo and Dr. Charles H. Mayo, founders of the Mayo Clinic. Sir Henry was a boyhood friend of the Mayo brothers, and this friendship was continued during his life.

The senior Dr. Mayo encouraged Sir Henry to study pharmacy and later arranged for his matriculation at the Chicago College of Pharmacy. During his attendance the Chicago Fire destroyed the college, and he then enrolled at the Philadelphia College of Pharmacy. At the age of twenty-one he was graduated as a member of the Class of 1874—in the days of Proctor, Maisch, Bridges and Remington.

Following his graduation from the Philadelphia College of Pharmacy, Sir Henry spent a few years in the retail drug business in New York. Later he took a position with the firm of McKesson and Robbins, and as their representative traveled extensively throughout the United States and Mexico.

Sir Henry left New York for London, and in 1880 with the late Silas M. Burroughs established the firm of Burroughs Wellcome & Co., manufacturers of fine chemicals and galenicals. Mr. Burroughs died in 1895, and since that time Sir Henry served as the head of Burroughs Wellcome & Co. In addition to the London organization, the firm has establishments in the United States, Italy, Canada, Australia, India, China, and other countries.

Sir Henry's American interests were wide and varied. He had been a life member of the American Pharmaceutical Association since 1875, and always took an active interest in its scientific work. In 1934 he was the Honorary President of the American Pharmaceutical Association and also was awarded the Remington Honor Medal for his scientific and other valuable contributions to pharmacy. In 1934 he received the honorary degree of Doctor of Science conferred by Marquette University, Milwaukee, Wisconsin.

As a result of Sir Henry's experience and interest in tropical research, Secretary of War, the Hon. J. M. Dickinson, appointed him to visit Panama and make a survey of sanitary conditions and methods of operation in all sections of the Canal Zone, and to submit an unbiased report based on his personal observations. The report of Sir Henry's survey secured a free hand for General Gorgas in continuing his monumental sanitary work in Panama.

Sir Henry was one of the sponsors and a director of the Gorgas Memorial Institute of Tropical and Preventive Medicine in Washington, operating scientific laboratories at Panama for research work touching causes and prevention of tropical diseases.

Sir Henry received world-wide recognition for his great services and princely contributions to science and medicine, for his interest in missionary enterprises and for his personal work in medical re-

search and in the history of medicine and for his archaeological and ethnological explorations and studies. His scientific achievements range from pioneering and the use of aerial photography in the making of archaeological surveys to the establishing of a number of research institutions.

During the World War, Sir Henry placed his scientific institutions at the service of the British Government. He instituted a Commission to improve design and construction of army ambulances. For use in Palestine and Egypt during the war, he constructed, equipped and supplied for the British Army Medical Service a chemical and bacteriological motor field laboratory. It was at this period that he became a British subject by naturalization.

In 1928, the honorary degree of Doctor of Laws was conferred upon him by the University of Edinburgh. In recognition of his life's work and generous support of medical research, he was knighted by the late King George V in 1932, and in 1936 was awarded the Croix d'Officier de la Legion d'Honneur by the French Republic. In 1936 the Spanish Republic awarded him the decoration of Comendador de la Orden de la Republica in recognition of his outstanding services to Spanish interests.

Apart from the experimental research laboratories of the establishments of Burroughs Wellcome & Co., which have to their credit an immense number of important original researches, Sir Henry has established a number of scientific institutions which are co-ordinated and under separate and distinct direction, including the following:

The Wellcome Physiological Research Laboratories—London—(1894)

The Wellcome Chemical Research Laboratories—London—(1896)

The Wellcome Bureau of Scientific Research —London — (1913) and the Museum of Medical Science (including Tropical Medicine and Hygiene, 1914) and the Auxiliary Entomological Research Laboratory at Claremont, Esher, Surrey—(1915)

The Wellcome Tropical Research Laboratories — Khartoum, Anglo-Egyptian Sudan, Upper Nile, Africa—(1901) and the fully equipped Auxiliary Floating Tropical Research Laboratory on the Upper Nile, and its tributaries—(1906)

As an energetic and public-spirited man, Sir Henry held memberships in numerous medical, archaeological, geographic and similar societies. He also received many honorary degrees in recognition of his scientific achievements and public benefactions.

NOTICES

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY

The next written examination and review of case histories of group B applicants by the American Board of Obstetrics and Gynecology will be held in various cities in the United States and Canada on Saturday, November 7, 1936.

Application blanks and booklets of information may be obtained from Dr. Paul Titus, Secretary, 1015 Highland Building, Pittsburgh (6), Pennsylvania. Applications for this examination must be filed in the Secretary's Office sixty days prior to the scheduled date of examination.

FISKE FUND PRIZE ESSAY
1937

The Trustees of the Fiske Fund announced at the annual meeting of the Rhode Island Medical Society, held in June 1936, that they proposed the following subject for the year 1937—"Newer Methods of Prevention and Treatment of Acute Anterior Poliomyelitis."

For the best essay on the subject worthy of a premium they offer the sum of two hundred and fifty dollars (250.00). Every competitor for the premium is expected to conform with the following regulations, namely:

To forward to the secretary on or before the first day of May, 1937, free of all expense, a copy of his dissertation with a motto thereon, and also accompanying it a sealed envelope having the same motto inscribed on the outside, and his name and address within.

Previously to receiving the premium awarded, the author of the successful dissertation must transfer to the Trustees all his right, title and interest in and to the same, for the use, benefit and behoof of the Fiske Fund.

Letters accompanying the unsuccessful dissertations will be destroyed unopened, by the Trustees, and the dissertations may be procured by their respective authors if applications be made therefor within three months.

The essays must be typewritten and should not exceed 10,000 words. If an essay be illustrated, such illustrations will be published at the expense of the author.

John E. Donley, M.D., Providence, R. I.; Walter C. Rocheleau, M.D., Woonsocket, R. I.; James W. Leech, M.D., Providence, R. I.—Trustees.

Wilfred Pickles, M.D., Secretary to the Trustees, 184 Waterman Street, Providence, R. I.

THE CANCER INSTITUTE

The first Cancer Institute will meet on September 7, 8 and 9 at the University of Wisconsin. Dr. William D. Stovall, director of the State Laboratory of Hygiene at the university, is chairman of the committee in charge.

Investigators from abroad who will speak at the general sessions and who will lead round table discussions include: Dr. Liev Kreyberg, of the University of Oslo; Professor Henry Coutard, chief of the department of x-ray therapy for cancer of the Radium Institute at the University of Paris, and Dr. Madge Thurlow Macklin, associate professor of histology and

embryology at the University of Western Ontario.

Americans who are expected to present papers are: Dr. C. C. Little, director of the Roscoe B. Jackson Laboratory for Cancer Research at Bar Harbor, Me.; Dr. Edgar Allen, chairman of the department of anatomy of the Medical School of Yale University; Dr. H. B. Andervont, biologist of the U. S. Public Health Service at Boston, Mass.; Dr. S. P. Reimann, pathologist and director of the Research Institute of the Lankenau Hospital and professor of experimental pathology in the Graduate School of the University of Pennsylvania; Dr. Emil Novak, associate gynecologist at the Johns Hopkins Medical School; Dr. J. B. Murphy, director of cancer research at the Rockefeller Institute for Medical Research; Dr. James Ewing, professor of oncology at the Cornell University Medical School, member of the staff of the Memorial Hospital for the treatment of cancer, New York; Dr. Gioacchino Fallia, physicist at the Memorial Hospital, and Dr. Warren H. Lewis, of the department of embryology of the Carnegie Institution of Washington, at Baltimore.

The expenses of the conference are being defrayed by the Wisconsin Alumni Research Foundation.—*Science*, August 7, 1936.

HARVARD UNIVERSITY TERCENTENARY CELEBRATION

SCHOOL OF PUBLIC HEALTH

Attention is called to the previous publication on page 1166 of the June 4 issue of the *Journal*, of the details of the program of the Harvard University School of Public Health. The Symposium on the Environment and Its Effect Upon Man will be held August 24 to 29, at the School of Public Health, 55 Shattuck Street, Boston.

REMOVALS

JOHN P. MONKS, M.D., announces the removal of his office from 264 Beacon Street to 330 Dartmouth Street, Boston. Telephone: Commonwealth 4314.

HARRY B. LEVINE, M.D., announces the removal of his office from 471 Commonwealth Avenue to 483 Beacon Street, Boston. Telephone: Kenmore 8000.

REPORTS AND NOTICES OF MEETINGS

MIDDLESEX NORTH DISTRICT MEDICAL SOCIETY

The quarterly meeting of the Middlesex North District Medical Society was held at the Vesper Country Club July 29, 1936, and the Moses Greely Parker luncheon was served to seventy Fellows.

Doctors Huber and Wakefield representing the State Department of Public Health explained to the Councilors the plan for carrying out the project for services to crippled children, which is to be financed from funds provided by the Social Security Act.

The subject is to be referred to a special committee, which will consult with Doctors Huber and Wakefield at a later date.

Dr. Tighe gave a complete report for the Public Relations Committee. The meeting showed that the younger men intend to be active in the work of our Society, which augurs well for the future.

Dr. Charles Mongan and Dr. Alexander Begg, President and Secretary of the State Society respectively, gave interesting talks on some of the problems of the Society, which were well received. The meeting was unusually large for our summer meeting.

FREDERICK P. MURPHY, *President*.

THE SOUTHWESTERN MASSACHUSETTS ASSOCIATED BOARDS OF HEALTH

The annual meeting of the Southeastern Massachusetts Associated Boards of Health was held at Fort Phoenix, Fairhaven, on Wednesday, July 29. The result of the election was the following list of officers for the coming year:—

President, Dr. T. L. Swift, Falmouth.

Vice Presidents, Joseph Christie, Dartmouth; Harold T. Cleveland, Dartmouth.

Secretary-Treasurer, George F. Crocker, Jr., Marstons Mills.

Executive Committee, Dr. R. P. MacKnight, New Bedford, F. W. Delano, Fairhaven, and Dr. W. O. Hewitt, Attleboro.

Routine business was transacted, but no papers were presented. There was the usual clambake.

HARVARD MEDICAL SOCIETY

The last meeting of the Harvard Medical Society for the current year was held on May 12, 1936, at the Peter Bent Brigham Hospital, Dr. George B. Wislocki presiding.

The medical case of the evening was presented by Dr. William Clauser: A sixty-nine year old white female entered the hospital three and a half weeks previously, complaining of frequent attacks of severe pain in the precordial region. Her family history was of interest, since practically all members in her own generation had died of arteriosclerosis and heart disease. One member of the family had died, at the age of eighty, of diabetes. Her past history was negative, and there was no history of diphtheria or any disease involving the heart. She had been well until four and a half years previously at which time, during an examination for minor complaints, her blood pressure had been found to be 190 mm. of mercury. Two and a half years previously she fainted, fell and injured herself. Except for extrasystoles no abnormalities were found in the cardiovascular system. She was next seen two years ago, at which time electrocardiographic studies showed bundle branch block. Her pulse rate at that time was 140 beats per minute. She was digitalized, and continued to take digitalis

until the present time. Nine months ago she experienced an attack of fainting and was extremely weak following it, remaining in bed for three months. Two and a half weeks before entry to the hospital she began to experience coldness of the hands and feet, and frequent fainting attacks. The frequency of the latter increased, and she experienced ten a day at the time of admission. Physical examination showed pale cyanotic extremities moist with perspiration. The radial pulse varied between fourteen and thirty beats per minute. An electrocardiogram showed regular auricular and ventricular rates, with complete dissociation of the two. She was given fifty milligrams of ephedrine every four hours, which elevated the pulse rate, and made her comfortable, until she began to experience attacks of ventricular tachycardia accompanied by fainting. The dose of ephedrine was decreased, which eliminated the tachycardia and fainting spells. The patient was presented as a case of complete heart block.

Dr. Henry A. Christian pointed out that symptoms were often the same whether the heart beat too fast or too slowly. Dr. Samuel Levine stated that there was probably some relation between Adams-Stokes' disease and diphtheritic infection at an early age. There is also some relation between this condition and gallbladder disease. If it is possible to rule out acute coronary disease, many cases of Adams-Stokes' disease will be found to have a history of one of the above conditions. In patients such as the one presented it is important to keep the heart rate regular at about thirty beats per minute. Too much ephedrine will precipitate ventricular fibrillation, and too little will allow the ventricle to beat too slowly.

The surgical case was presented by Dr. R. L. Peterson: A fifty-five year old white housewife entered the hospital three weeks previously complaining of vaginal and rectal bleeding of one year's duration. The menopause had occurred ten years previously, and except for slight leucorrhea of two years' duration there had been no vaginal discharge until the onset of the present illness. Three years ago she experienced "heart attacks" and dyspnea, and had been treated with digitalis. One week before admission she began to notice a tingling sensation and a feeling of numbness in her left hand. Physical examination on entry revealed cyanosis and coldness of the fingers of the left hand. The radial pulse was not palpable, although brachial pulsations were felt at all times. The blood pressure in the left arm was 140/70 mm. of mercury. X-ray examination showed that there was no cervical rib. Pelvic examination showed a large friable mass in the cervix with multiple implants on the vaginal walls. A biopsy of this mass proved it to be a carcinoma, and 3000 milligram hours of radiation therapy were given. The left hand became progressively colder and more cyanotic, and the brachial artery was explored at its bifurcation. No obstruction was found, although the pulsations were of poor quality. It was decided that there was some obstruction higher in the arterial system of the left arm. Treatment with alternate pressure and suction to the left hand and forearm was instituted with some improvement.

Dr. Elliott C. Cutler stated that he believed that the symptoms in this case were caused by a "silent embolus" in the axillary artery.

The paper of the evening was delivered by Florence R. Sabin, M.D., Member of the Rockefeller Institute for Medical Research, New York City, who spoke on the subject "Development of the Cells of the Blood and Bone Marrow." Dr. Sabin's studies were carried out on embryos and young rabbits, comparative counts of the various forms of cells in the blood and bone marrow being made. In fetal life the hemoglobin is higher in percentage than would be expected from the number of red blood cells present in the blood stream. There is thus a sort of "primary anemia" present, which is not corrected until the third month of extra-uterine life. Dr. Sabin interprets this as indicating that the very young rabbit can produce hemoglobin with ease, but that its ability to form new reticulocytes is imperfect until the "extrinsic" and "intrinsic" factors come into play.

Shortly after the birth of the animal there is a "flooding" of the blood stream with large numbers of immature red blood cells, chiefly reticulocytes, which are derived from the liver. Then during the first week of life there is a true fall in the number of circulating red blood cells as indicated by a drop in the reticulocyte count. At the end of this time there is a rapid increase in the erythrocytes, and the normal adult level is reached by the end of the third week of life.

This rise in the number of red blood cells takes place during the time that there is an extremely rapid growth of bone and bone marrow. Counts of the marrow cells (using the supravital staining technique) showed that members of the red blood cell series were in predominance during the first week of life, but these could not be passed out into the blood stream because they were not yet mature. They become mature during the second week of life, the time when the red count in the peripheral blood stream begins to rise.

There are two theories as to the exact site of origin of the red cells in the marrow. One school holds that they are formed extravascularly, the other that they originate and grow within the sinusoids. Evidence is not conclusively in favor of either theory, but Dr. Sabin has demonstrated microscopically many groups of immature red cells lying within the sinusoidal walls. No one has yet proved that the red cell has any power of motility, which would enable it to enter the vascular system, if it did originate extravascularly. These two facts point toward an intravascular origin of the erythrocyte.

The monocytes reach their full quota in the blood during the first week of life, but the neutrophiles are held down during the whole first month of life by the "drive for red cells". The lymphocytes rise rapidly during the first month to their normal level.

By means of supravital staining of marrow from fetal animals, Dr. Sabin has been able to identify a "primitive cell," which she believes is the parent from which both the red and white cell series are derived. In very young embryos this cell is the only one found in the marrow, but at birth it composes only 15 per cent of the total, and in later life falls to but 5 per cent of the total. In the pathological conditions of agranulocytosis and combined anemia and leucopenia there is an absolute increase in the number of these primitive cells present.

A detailed study of the monocytic series has convinced Dr. Sabin that monocytes are to be regarded as a more youthful form of the macrophage, which it becomes with increase in its age. Both cells have many points of similarity between them, e.g., active phagocytosis, and motility by means of a "veil".

Dr. Sabin has studied the phagocytic properties of monocytes, macrophages, and epithelioid cells by observing their reactions to injections of waxes, tuberculoproteins, complex polysaccharides, and phosphatides. Waxes are taken up by cells of the monocytic series, which fuse and form giant cells. After a rather prolonged period these giant cells are able to disperse the wax, and dispose of it. Tuberculoprotein is phagocyted in a similar fashion, and stimulates the production of monocytes and a few epithelioid cells. The phosphatide gives a pure reaction of epithelioid cells and causes the production of a typical tubercle. The tuberculopolysaccharide after intraperitoneal injection calls leucocytes from the blood stream, and so damages them that they are subsequently phagocyted by the macrophages of the "milk spots" of the omentum. (The "milk spots" are accumulations of monocytes and macrophages with an occasional "primitive cell".) These leucocytes are absorbed in the vacuole of the macrophage by an enzymotic process. As a result of these studies, Dr. Sabin has concluded that the epithelioid cell should be considered as a cell that has phagocyted some material which it cannot dispose of, due to the fact that it is not physiologically adapted to break down very complex molecules. These cells eventually die, forming a caseous mass, which in turn may be phagocyted by other epithelioid cells.

Dr. Sabin pointed out that the fall in lymphocytes in the blood stream which occurs in cases of tuberculosis, syphilis and cancer parallels loss of weight and failing resistance. During this same time there is a rise in the number of monocytes.

WORCESTER DISTRICT MEDICAL SOCIETY
1936-1937

Wednesday, September 16, 1936 — The Milford Hospital, Milford, Mass.

4:30 p. m. Visitation of the Milford Hospital unit, which has been recently refinished and added to.

6:15 p. m. Dinner — complimentary by the hospital.

7:30 p. m. Scientific program and business session. Speakers and subjects to be announced later.

Wednesday evening, October 14, 1936—Rutland State Sanatorium, Rutland, Mass.

6:15 p. m. Dinner — complimentary by the State Hospital.

7:30 p. m. Business session and scientific program. Speakers and subjects to be announced in a later issue of the *Journal*.

Wednesday evening, November 11, 1936 — Grafton State Hospital, North Grafton, Mass.

6:15 p. m. Dinner — complimentary by the hospital.

7:30 p. m. Business session and scientific program.

Wednesday evening, December 9, 1936—St. Vincent Hospital, Worcester, Mass.

6:15 p. m. Dinner — complimentary by the hospital.

7:30 p. m. Business session and scientific program.

Wednesday evening, January 13, 1937 — Worcester City Hospital, Worcester, Mass.

6:15 p. m. Dinner — complimentary by the hospital.

7:30 p. m. Business session and scientific program.

Wednesday evening, February 10, 1937—Worcester State Hospital, Worcester, Mass.

6:15 p. m. Dinner — complimentary by the hospital.

7:30 p. m. Business session and scientific program.

Wednesday evening, March 10, 1937—The Memorial Hospital, Worcester, Mass.

6:15 p. m. Dinner — complimentary by the hospital.

7:30 p. m. Business session and scientific program.

Wednesday evening, April 14, 1937 — Worcester Hahnemann Hospital, Worcester, Mass.

6:15 p. m. Dinner — complimentary by the hospital.

7:30 p. m. Business session and scientific program.

Wednesday afternoon and evening, May 12, 1937—Annual Meeting. Time and place for this meeting will be announced in an early spring issue of the *Journal*.

ERWIN C. MILLER, M.D., *Secretary*.

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, AUGUST 17, 1936

Wednesday, August 19—

†12 m. Clinico-Pathological Conference. Children's Hospital.

Saturday, August 22—

*10 a. m. - 12 m. Staff Rounds at the Peter Bent Brigham Hospital. Conducted by Dr. Reginald Fitz.

*Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

August 24-29—Harvard University Tercentenary Celebration. See page 1166, issue of June 4. School of Public Health, page 310 of this issue.

September 4 to 8—First Congress of the Austrian Society for the Study of Roentgenology. For details address: Allgemeines Krankenhaus, Alserstrasse 4, Wien, IX, before September 1.

September 7, 8, and 9—The Cancer Institute. See page 309.

September 7-10—International Union against Tuberculosis. See page 554, issue of March 12.

September 7-11—American Congress of Physical Therapy will meet at the Waldorf-Astoria, New York City. See page 52, issue of July 2.

September 9 to 12—Sixth Congress of the International Society for Urology. For details address Dr. Theodor Hrynteschak, Rathausstrasse 3, Wien, I.

September 14 and 15—Tercentenary Session of the Harvard Medical School. See page 1166, issue of June 4.

September 16-21—First International Congress of Sanatoria and Private Nursing Homes. See page 803, issue of April 16, and page 264, issue of August 6.

September 22, 23, 24—Twelfth Clinical Congress of the Connecticut State Medical Society. See page 217, issue of July 30.

October 12-18—Third International Congress on Malaria. See page 1076, issue of May 21.

October 19-23—Clinical Congress of the American College of Surgeons. See page 180, issue of January 23.

October 19-31—1936 Graduate Fortnight of the New York Academy of Medicine. See page 1221, issue of June 11.

October 20-22—Academy of Physical Medicine, Annual Meeting, Hotel Statler, Boston.

October 20-23—The American Public Health Association. See page 1226, issue of June 11.

March 30 - April 2, 1937—First International Conference on Fever Therapy. Postponement notice. See page 52, issue of July 2.

April 21-24, 1937—American Society for Experimental Pathology. See page 1075, issue of May 21.

DISTRICT MEDICAL SOCIETY

WORCESTER DISTRICT MEDICAL SOCIETY

September 16, 1936 - May 12, 1937—See page 312.

BOOK REVIEWS

Handbook of Bacteriology. For Students and Practitioners of Medicine. Joseph W. Bigger. 458 pp. Fourth Edition. Baltimore: William Wood & Company. $4.25.

The author has attempted to present the sciences of bacteriology and immunology in a small text of 443 pages—an impossible task. Too much of the book is devoted to a description of simple techniques (fully a page is occupied with a description of how to focus a microscope). Such procedures have received abundant description already and are best taught students by laboratory instructors.

The subject matter is presented in a general manner without detail. For this reason the book is a broad, descriptive outline. Much is lacking in the way of useful, detailed information, a fact which detracts from its value for medical students, the medical profession, public health officers and laboratory workers.

The information given is accurate and is presented in a simple, clear style. It should be an excellent text for a general course in bacteriology for undergraduate college students and nurses. It is not adequate for more comprehensive courses or for courses intended to prepare students for actual work in bacteriology. There are a number of similar texts of equal merit on the market at the present time.

La Rate en Pathologie Sanguine. E. Houcke. 154 pp. Paris: Masson et Cie. 45 fr.

The present monograph contains the author's results of ten years' research on the pathologic anatomy of the spleen. For the most part these were conceived with the changes that occur in this organ in pathologically altered blood states. The work is divided into four parts. The first subdivision deals with the changes in the spleen in the various leukemias. Also included in this section are studies of Vaquez's disease and tuberculosis of the spleen. The second portion is composed of descriptions of the splenomegalies accompanying reticulo-endothelial changes, Gaucher's disease, leukemia with monocytosis and lymphogranulomatosis. The third division contains observations of changes occurring in connection with pernicious anemia and hemolytic icterus. The last chapter is devoted to circulatory disturbances of the spleen, particularly splenic infarct and Banti's disease. An extensive bibliography and a number of excellent photomicrographs accompany the text. The book is a distinct contribution to either the consideration or study of the spleen and should prove of particular value to the pathologist and the hematologist.

Clinical Diagnosis by Laboratory Methods. A Working Manual of Clinical Pathology. James Campbell Todd and Arthur Hawley Sanford. Eighth Edition, Thoroughly Revised. 792 pp. Philadelphia and London: W. B. Saunders Company. $6.00.

The eighth edition of this standard text will be welcomed by laboratory workers throughout the country. The growth of a specialized field of clinical pathology is well attested by the comparison of this with the first edition, which was one of the pioneers in the field published in 1908. This handbook has the advantage of presupposing very little experience on the part of the technician, so that the tests are outlined in much detail and all its steps clearly given. The procedures are well selected and generally recognized as standard. Those experienced in this field will recognize that it is a guide and handbook rather than a reference book. For the practitioner who wishes to keep his hand in at a little laboratory work, and for the inexperienced technician, it will be of great value. Illustrations are numerous and clear.

The Next Hundred Years. The Unfinished Business of Science. C. C. Furnas. 434 pp. Baltimore: The Williams & Wilkins Company. $3.00.

A visit to the recent Century of Progress resulted in this book. Pointing to the dubious nature of much of our "progress," the author traces from Biology to the Social Consequences the results of much scientific endeavor and charts future developments. Few

facets of life have been omitted in this provocative work, worthy of the best efforts of such a noted leader as C. C. Furnas. With the collateral reading suggested, one could profitably spend many months following indicated leads. Facility and lucidity of expression characterize humorous suggestion intermingled with restraint that make the work doubly interesting, holding the reader to the very last. One can but agree with Furnas when he deplores the narrow specialization of today; yet the average individual, unless endowed with more than normal capacity, can hope to cover but small parts of the enormously wide field of his own work. To the doctor still interested in the range of science beyond the bread-and-butter type, the reviewer earnestly recommends this very readable work.

Glandular Physiology and Therapy. A Symposium Prepared Under the Auspices of the Council on Pharmacy and Chemistry of the American Medical Association. 528 pp. Chicago: American Medical Association. $2.50.

This is a reprint of the series of papers which appeared in the *Journal of the American Medical Association* last year. Usually such a compilation of multiple authorship is difficult to read and of uneven quality. This is remarkable in the uniform excellence and thoroughness of presentation. On account of the frequent references to the original literature, these summarizing chapters vary less in style even than such collections are wont to do. It is impossible adequately to review each chapter but the authoritativeness of presentation makes one recommend the book unhesitatingly as essential to every physician, as there is no field of the practice of medicine which has been left untouched by the recent advances in endocrinology.

Diseases of the Liver, Gall Bladder, Ducts and Pancreas, Their Diagnosis and Treatment. Samuel Weiss. 1099 pp. New York: Paul B. Hoeber, Inc. $10.00.

It is possible that a consideration of diseases of the liver and biliary system demands a volume of almost 1100 pages. The first impression in reading this enormous compendium is amazement at the erudition of the author. There is no doubt that the book contains a tremendous amount of information as is evidenced by the fact that references alone cover over ninety pages of closely printed matter. It is probable that this reference list which covers a multitude of articles on all subjects germane to diseases of the biliary tract constitutes the greatest contribution to the book. The actual subject matter of the book, however, is beyond the scope of any reader except for purposes of reference and even here the material is frequently presented in a confusing and poorly correlated manner. The history, physiology, pathology and general discussion of diseases of the biliary tract is presented in amazing detail. The vast majority of the facts are correct but many of the details are presented with no critical thought and a rather careless arrangement. Certain important omissions can readily be noted. Altogether too much optimism and faith is put upon the diagnostic and therapeutic value of duodenal drainage. Therapeutic measures for various forms of liver diseases are complicated, too diversified and at times do little but confuse the reader. The consideration of such a common disease as catarrhal jaundice is poorly planned and does not take into account the more recent physiological and pathological findings in relation to the disease. Symptom complexes and clinical entities are intermingled in such a way as to leave the reader in doubt about given conditions, and contradictions and partial contradictions are frequent. Certain of the discussions, particularly the anatomic considerations of biliary tract disease, are excellent. The discussion of cirrhosis is on the whole fairly satisfactory but it is too complicated. Jaundice as a symptom is well described but poorly discussed. Treatment is based too much upon diversified methods more frequently seen on the Continent than in this country and not enough emphasis is placed upon direct methods indicated by clinical experience in given liver conditions.

There is practically nothing in the book that is simple and straightforward and no attempt has been made to clarify the conception of a sick liver. There seems to be an attempt to impress by completeness, although the book is far from complete. It represents a sort of mausoleum in which are buried all sorts of information and some misinformation. On the whole it does not form a real contribution to medical literature and it is to be regretted that the vast clinical experience of the author was not utilized as the basis of a much simpler volume, condensed into smaller space with a direct approach to many of the difficult problems connected with diseases of the biliary tract.

Traité de Physiologie Normale et Pathologique. Published under the direction of G. H. Roger et Léon Binet. Tome X. Fascicule II. 1579 pp. Paris: Masson et Cie.

With the tenth volume, this treatise dealing with the normal and pathologic physiology of every system of the human body, is complete. The entire work is comparatively vast and comprehensive in scope and ably portrays the advanced state of French physiology. The last two books continue the format of those that have previously appeared, and like their predecessors, completely cover the field dealt with. In addition to the presentation of the accepted phases of the physiology of the nervous system, there are included the results of much research. There are excellent chapters on the physiology of the skin by G. Milian, the physiology of hearing by A. Strohl, the sympathetic and parasympathetic systems by a number of contributors and the cerebrospinal fluid by J. Haguenau. The final volume is well conceived and excellently written and is highly recommended to the physiologist, the neurologist and the internist.